ARCHIVES of OPHTHALMOLOGY

VOLUME 15 (old series Volume 72) APRIL 1936 NUMBER 4

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FUNCTIONAL DISTURBANCES OF THE EYES

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The functional disturbances of the eyes which I wish to discuss here may be caused either by anomalies of the dioptric or of the motor apparatus or by anomalies the nature of which is not yet known. Though they have been discussed many times, they are not fully understood at present. For instance, it is not known why various subjective disorders, such as eyestrain and headache, may result from comparatively slight degrees of heterophoria or astigmatism, whereas the majority of patients, even those with a high degree of the same anomalies, do not complain of any discomfort. That there is, indeed, a causal connection between the subjective disorders and the anomalies under discussion is evidenced by the frequent disappearance of the symptoms after a proper correction of the anomalies. One of the characteristics is the similarity of the subjective symptoms of which patients with different anomalies complain. Eyestrain, headache, rapid fatigue and blurring in reading, dizziness and nausea are complained of by patients with refractive errors as well as by those with heterophoria or aniseikonia, so that subjective symptoms, as a rule, do not offer valuable suggestions as to the differential diagnosis. Nevertheless, the history obtained from the patients is rather important. Since many of them show a neuropathic constitution, an inherited disposition should be looked for. If questioned, some patients may give definite statements as to the onset and possible cause of their disturbances, i. e., psychic shock or physical exhaustion after a period of hardship, as was frequently noted during the war, or as the result of a severe illness. One may obtain some clues as to the origin of the subjective disorders by ascertaining whether they have developed gradually or have arisen suddenly. The objective examination must precede the subjective tests. Retinoscopy and ophthalmometry will disclose a possible refractive error, the correction of which by means of glasses may improve the visual acuity in many cases. The other disturbances if caused by the refractive error will disappear promptly.

Read before the Brooklyn Ophthalmological Society, Brooklyn, N. Y., Dec. 19, 1935.

Eyestrain, headaches and other symptoms are met with in cases of hyperopia or astigmatism much more frequently than in cases of myopia, since they are probably due either to an excessive or to a continually varying accommodation, the latter particularly in cases of astigmatism. The patient tries, without succeeding, to obtain a sharp retinal image by alternately strengthening and weakening the innervation of the ciliary muscles. So-called accommodative asthenopia develops mostly in consequence of physical and psychic exhaustion. It is not unusual for a patient to require glasses when convalescing from an illness but to put them away after full recovery. If in a patient with a comparatively small refractive error the disorders do not disappear after an exact correction, another anomaly must be considered as the main cause of the difficulties.

Careful statistics have proved that heterophoria occurs just as frequently as ametropia, namely, in more than 80 per cent of cases. That is to say, neither heterophoria nor ametropia must be considered unconditionally as a pathologic condition, although textbooks formerly identified emmetropia with normal refraction, just as orthophoria was considered the normal position of the eyes. Both should be called "ideal" conditions (from a didactic point of view), since they present the starting point for the discussion of ametropia and heterophoria. Orthophoria is to be understood as that condition in which the visual lines as well as the corresponding retinal meridians are parallel if the eyes are directed to a distant point and if the motor innervation is completely relaxed. In that case the position of the eyes depends solely on mechanical factors, i. e., on the topographic-anatomic relations between the eyeballs and their adnexa; on the physical qualities of the latter, their elasticity, volume and so forth, and finally on the size and shape of the orbits. Orthophoria, though theoretically a definite condition, rarely occurs, since the physiologic asymmetry of the face does not admit a perfect identity of the orbits and of the intra-orbital tissues which determine the position of rest of the eyes. Thus, one must consider heterophoria, though it differs in one way or another from the ideal type, as the rule and orthophoria as the exception. This has been substantiated by clinical statistics. Heterophoria has been found in at least 80 per cent of thousands of healthy persons of different ages and both sexes, with approximately emmetropic refraction and without any subjective disorders. Still more convincing have been the results of careful investigations of persons who, either through injuries or disease, have acquired amaurosis or such a high degree of amblyopia of one eye that binocular vision has been abolished.[1] In practically all these cases strabismus of different kinds and degrees develops. In the majority

1. Bielschowsky, A.: Ueber die relative Ruhelage der Augen, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **12**:67, 1913.

divergent squint develops, especially in adults. Convergent strabismus is found after the loss of binocular vision five times as frequently in children up to 15 years of age as in persons of 30 or more years, a fact to be explained by the well founded assumption that in adolescence there is a gradual change of the topographic-anatomic factors, such as produce a more divergent or a less convergent position of the eyes. Both horizontal deviations are frequently combined with a vertical and a rotary component. The results obtained by the aforementioned statistical investigations are important, since they present the only definite proof of the frequency of the different kinds of heterophoria.

As I always test the motor apparatus of my patients regardless of the disorders for which they consult me, I have observed that in numerous cases squint of a considerable degree develops after the loss of binocular vision, although when the patient is first seen only a slight amount of heterophoria, if any, can be determined. That can be explained only by assuming that the usual tests for heterophoria do not completely relax the fusion innervation, so that the whole amount of the latent deviation becomes manifest, which would be the same as disclosing the anatomic position of rest. If this task could be accomplished it would be easier to decide on the kind of treatment, for it depends in many cases on the position of rest. In cases of heterophoria one cannot consider a muscle operation which would change the anatomicomechanical condition before one has made sure what part may possibly be played as an etiologic factor by abnormal innervation.

It is practically impossible to eliminate all the types of innervation that influence the position of the eyes. Even in sleep or in a deep narcosis there are almost constant movements of the eyes in parallel, opposite, lateral or vertical directions. The voluntary movements, of course, can be eliminated by ordering the patient to look straight forward at a distant point during a test while any possible hyperopia causing an accommodative effort which might involve the convergence innervation is corrected. By eliminating the movements of the head and of the body the vestibular innervation, based on the connections between the vestibular and the oculomotor centers, also can be eliminated. Other types of innervation, the origin of which is still unknown and which cannot be excluded by any means, frequently play an important part in producing unsteady vertical deviations; they will be discussed later. But the main difficulty encountered in testing for heterophoria comes from the powerful fusion apparatus which conceals the anomalous position of rest. The fusion apparatus may be compared with a reflex mechanism. If the images of one outlying object are formed on disparate, i. e., noncorresponding, retinal points, the position of the eyes relative to each other will be altered in such a way that the images will be shifted in corresponding areas and fused. Fusion innervation,

as a rule, takes place automatically, but it is not a true reflex. While fusion occurs at its best in a state of inattention, fusion movements are prevented if the attention is diverted. The visual stimuli are conveyed to the cortex and come into consciousness in order to produce a motor innervation, whereas the pathways for true reflexes are subcortical. In a subject with orthophoria the only function of the fusion apparatus is to produce an increase and decrease of convergence according to the distance of the object attracting attention. The other types of fusion innervation take place in persons with heterophoria, and they prevent an anomalous lateral, vertical or rotary deviation from becoming manifest.

It is known from wide clinical experience that the fusion innervation does not relax completely after the fusion tendency has been broken. If tests for heterophoria are repeated several times under apparently like conditions, the results obtained in the tests frequently differ, not only with respect to the amount but occasionally also with respect to the kind of existent heterophoria. The double images in the second test may be found to be farther apart or closer together than in the first or the third test, and in the fourth test they may display a vertical as well as lateral separation. This behavior proves that the tests are inadequate, not only because they do not bring about complete relaxation of the fusion innervation but also because they fail to produce partial relaxation of the same degree in repeated tests. The increase of the tonus of the muscles which are brought into play by the fusion tendency in order to overcome an anomalous position of rest is not reduced to the normal amount immediately after the fusion tendency is broken; it diminishes slowly but neither continually nor equally, and a residue of the surplus of tonus covering a fraction of the heterophoria may remain for a long time.

To understand this behavior one should produce an artificial heterophoria in oneself by placing prisms of increasing strength base down before the right eye until that prism is found which cannot be overcome by superduction. If the experiment is repeated several times in succession and the strength of the prisms is increased slowly, one will tolerate stronger prisms in the later experiments without seeing double, till at last the strongest prism will be found beyond which one cannot go without causing diplopia, however often the experiment may be repeated. If the prism is removed, diplopia corresponding to hyperphoria of the right eye appears for a few seconds, followed by binocular single vision. However, for about fifteen minutes after the removal of the prism a dark red glass held before one eye will uncover a fraction of the hyperphoria of the right eye produced in the prism test. As a rule, it takes from fifteen to thirty minutes for the orthophoria or the original type of heterophoria to return; and even then a small residue

of the hyperphoria of the right eye which was produced experimentally may return and be displayed by later repetitions of the red glass test, particularly if the prism has been worn for some hours or days. One learns from such an observation that the so-called compensating innervation brought into play during the experiment does not disappear immediately when the fusion stimulus ceases but that it decreases slowly and not always without a temporary recurrence of a fraction of the compensating fusion innervation. In the face of such behavior one will understand that a fusion innervation which has been necessary since early childhood to overcome an anomalous position of rest will be more resistant than any innervation produced by the prism experiment and that temporary suspension of binocular single vision will reveal only a fraction of the heterophoria, a greater or smaller fraction according to circumstances, the nature of which frequently cannot be explained satisfactorily.

Considering that with the usual methods of investigation the fusion innervation is not interrupted long enough to make the entire amount of heterophoria manifest, Marlow [2] recommended the prolonged occlusion test of one eye. He found in numerous cases that the angle of deviation increased in correspondence with the length of time the bandage was worn and that after a week the deviation was, as a rule, constant. But there are a few patients who then show a smaller degree of squint than was found before the bandage was applied, and some patients are seen in whom the kind of deviation at the end of the occlusion period is opposite to that noted before the occlusion. In a number of cases a different amount as well as a different kind of heterophoria becomes manifest if first one eye and then the other is occluded. Marlow noted, as a result of such an alternate occlusion performed in eighty-two cases, hyperphoria of the same kind but of a different degree in about 60 per cent; in more than 28 per cent there was also a different type, each eye when uncovered deviating upward. These observations prove that even if binocular vision is interrupted for several weeks it is not sufficient to abolish all the innervations by which the position of the eyes relative to each other is influenced.

There are, indeed, some types of innervation that have nothing to do with the fusion apparatus and that change the position of the eyes at irregular intervals. One type produces either unilateral or bilateral dissociated vertical movements. The latter type of movement is known as double or alternating hyperphoria. It was wrongly taken as a particular kind of heterophoria, being based on an imbalance between the elevator and the depressor muscles of the eyes, the former being con-

2. Marlow, F. W.: The Influence of Prolonged Monocular Occlusion in Revealing Errors of Muscle Balance, Brit. J. Ophth. **4**:145 (April) 1920.

sidered stronger. I have been able to prove by a thorough investigation of many hundreds of cases, the most characteristic features of which I described in a detailed publication in 1931,[3] that double hyperphoria is due not to muscular imbalance but to involuntary intermittent and alternating excitations of the centers of both positive and negative vertical divergence, which I call hyperphoria of the right and of the left eye, respectively. Only one phenomenon which is present in every case of double hyperphoria may be mentioned here as indisputable proof of the nervous origin of the anomaly. Each eye when covered moves upward. But if the eye behind the screen is observed while the other eye is fixed on a light, it will be seen that the screened eye, instead of keeping to a certain degree of deviation, is moving slowly up and down at irregular intervals, not as a rule, however, passing the horizontal plane in the downward direction. If a darkening glass wedge (Zeiss) is placed in front of the fixing eye and moved in such a manner that the fixed light is gradually darkened, it will be seen that the covered eye moves downward below the horizontal plane in nearly exact proportion to the darkening of the fixing eye.

From the evidence of the nervous origin of double hyperphoria one must infer that it does not belong to the true heterophorias, which are based on anatomicomechanical conditions, and it would therefore be a mistake to operate on a patient with pure double hyperphoria as if the condition were an anomaly of the position of rest. But double hyperphoria is frequently combined with some kind of latent or manifest horizontal deviation (I found it in more than 41 per cent of cases of heterophoria), and if one succeeds in correcting these deviations and obtaining fusion the double hyperphoria will become latent without causing any subjective disorder, since the fusion power overcomes the anomalous excitation of the vertical divergence centers.

It is obvious that the apparently contradictory results obtained in the same patient by Marlow's prolonged occlusion test, according to whether the right or the left eye was bandaged, may be attributed to anomalous types of innervation, such as vertical divergence innervation, altering the position of the eyes relative to each other if the fusion tendency is eliminated as well as when it is only weakened. Those involuntary and intermittent types of innervation may be responsible, at least in part, for the considerable differences so frequently observed in the usual tests for heterophoria if the same subject is examined several times under apparently similar conditions, although the variable and imperfect relaxation of the compensating fusion innervation must be considered as the more important factor to which the differences are to be attributed.

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3. Bielschowsky, A.: Die einseitigen und gegensinnigen ("dissoziierten") Vertikalbewegungen der Augen, Arch. f. Ophth. **125**:493, 1931.

There is, in my opinion, only one test that brings about at least an approximately complete relaxation of the fusion innervation which compensates the anomalous position of rest—the prism duction test. If the range of a certain fusion movement goes far beyond the average limits which are determined by the same kind and the same number of tests applied to subjects with orthophoria, whereas the range of the antagonistic fusion movement in the case under discussion is abnormally narrowed, it is fairly certain that there is an anomalous position of rest corresponding to the enlarged fusion movement. A patient with hyperphoria of the right eye, for instance, will be able to overcome prisms up to 10 prism diopters or more base down before the right eye or base up before the left, whereas the weakest prism in the reverse position causes insuperable diplopia. The hyperphoria of the right eye, or positive vertical divergence, as I call it, is kept latent under normal conditions of seeing by the compensating negative vertical divergence innervation. The latter is made to relax gradually by putting prisms of increasing strength base down in front of the right eye or base up in front of the left. Before the muscles governed by the positive vertical divergence innervation are contracted in order to overcome the increasing prism strength, the compensating innervation by which the hyperphoria is usually kept latent must be relaxed. If at the very moment of the appearance of diplopia the prism held before one eye is replaced by a dark red glass, the distance between the double images indicates the kind and approximate amount of the hyperphoria. That the range of abduction in cases of exophoria is frequently found to be enlarged without restriction of the range of adduction depends on the exceptional position of the convergence innervation. It is the only fusion innervation which, as a link of the mechanism for near vision, is governed to a certain extent by the will and, owing to constant use, is exercised much more than the other types of fusion innervation. Therefore, in cases of esophoria one cannot expect to obtain an approximate measurement by the adduction power test, though the restriction or the total lack of abduction power will indicate the presence of esophoria.

To make the best possible use of the duction power test in the investigation of heterophoria and to avoid mistakes, one must remember that all types of innervation governed by the will as well as by the fusion apparatus go without exception equally to the two eyes. This law, which was formulated by Hering, has been substantiated by clinical experience. Some writers have doubted that it is true and have cited the fact that some persons can move one eye alone or the two eyes unequally. These authors did not take into consideration that unilateral or unequal movement of the eyes must occur if two impulses are sent at the same time to the two eyes, as happens if, while the eyes are directed to a distant point, another point nearer and to the left

of the former one attracts the attention. Then two types of innervation are required: one for convergence and the other for turning the eyes to the left. Both impulses drive the right eye to the left, whereas the left eye is driven to the right by the convergence impulse and simultaneously to the left by the levoversion impulse. The result must be that the left eye, in spite of having the same innervation as the right eye, will move less than the latter or not at all.

Apart from other proofs furnished by Hering, the truth of this explanation follows from observations made in the duction power test. Suppose one has determined the limit of superduction of the right eye in a series of tests with prisms of increasing strength. If, immediately afterward, prisms in the reversed position, base up, are placed before the left eye, one will find that in the first test the same amount of infraduction of the left eye will be obtained at once which was obtainable only after a series of successive experiments when superduction of the right eye was tested. But if infraduction of the right eye is tested immediately after a series of superduction tests of the right eye, one will find that the patient can overcome only a weak prism or no prism at all. And just as little superduction of the left eye is he able to produce if examined immediately after a series of superduction tests of the right eye. This experience is explained as follows: In testing the superduction of the right eye a positive vertical divergence innervation is elicited, driving the right eye upward and the left eye downward. In order to maintain fixation a superversion impulse is required so that the left eye remains unmoved, whereas the right eye is driven upward by both types of innervation. After a series of positive vertical divergence tests an increased tonus of the right elevator and of the left depressor muscles is left, which does not disappear immediately if the prism is removed. So if one attempts to elicit the antagonistic negative vertical divergence innervation without the elapsing of a sufficient interval, the right depressor muscles and the left elevator muscles cannot overcome the resistance offered by the increased tonus of the antagonistic muscle groups. On the other hand, since the innervation required for infraduction of the left eye is the same positive vertical divergence innervation which is called into play by the superduction tests of the right eye, the same amount of infraduction of the left eye is brought about by the first experiment as was obtained in the last superduction test of the right eye.

The conclusions to be drawn from physiologic experiments and clinical experiences are as follows:

1. In making use of the prism duction tests for determining the position of rest the results will be reliable to a certain extent only if every duction movement is tested several times in succession, because the limits of fusion movements are not rigid and it depends only on

the modus procedendi of the experiment and on the time spent on it whether one finds, for instance, a superduction of 2 or of 5 degrees in a case of orthophoria. Antagonistic fusion or duction movements must not be tested in immediate succession, because the increased tonus of the muscle groups called into play by the first series of experiments would let the function of the antagonistic muscle groups appear weaker than it is actually found to be if the second test is made after a pause of about fifteen minutes or is repeated at least six times in succession until the strongest prism that the patient is able to overcome is determined. Only such a procedure gives a certain security that the innervation used to keep the heterophoria latent has relaxed ad maximum.

2. The single steps of a thorough investigation of heterophoria are as follows: One must start with an objective test. While the patient's eyes are directed straight forward to a little light forming the center of the Maddox tangent scale at a distance of 20 feet (6 meters), one covers each eye alternately, observing, after the screen has been taken away, whether the eye moves, and if so in which direction, in order to achieve fixation. From this the kind and the amount of such movement can be estimated, and it can be learned besides whether the answers of the patient are reliable in the following subjective tests for measuring the deviation. For inhibiting the fusion tendency a dark red glass held before one eye will be sufficient in most cases. It must be dark enough to eliminate all retinal images except the image of the lamp in the middle of the Maddox tangent scale. If the patient is asked where he sees the red lamp, he will fix the lamp with the eye behind the red glass so that the red image lies in the fovea. In a case of exophoria or esophoria the red image is seen covering the number to which the visual line of the second eye is directed, according to the law that two images formed on a pair of identical points of the retinas are localized in one and the same visual direction; in a case of hyperphoria the red image is seen above or below the other. It should be noted particularly that in measuring a paretic deviation the eye behind the red glass is the fixing eye, so that the primary deviation is determined if the red glass is held before the normal eye and the secondary deviation is determined if the red glass is before the paretic eye. If the red glass alone fails to produce diplopia it may be combined with a prism of 10 prism diopters. The displacing effect of the prism held alternately base in or base down, first before one eye and then before the other, must of course be taken into consideration in the measurement of the deviation.

After having measured the deviation in the primary position of the head and of the eyes, first with the right and then with the left eye fixing, one must ascertain whether a change of the deviation is to be recorded in looking toward the different peripheral parts of the field

of fixation. Instead of moving the lamp fixed by the patient his head is turned so that he has to move his eyes in the opposite direction to keep the fixation unaltered. Even in a most careful investigation of a patient whose statements have been proved to be fully reliable, the measurements of the deviation found in various parts of the field of fixation will differ. Repetition of the examination will show whether the same differences return, following a certain rule, as, for instance, in a typical case of paresis, or whether no rule is to be ascertained, the amount as well as the kind of deviation varying incalculably. Slight differences of the latter type are not exceptions but the rule in cases of heterophoria. They are due, as has been said, in the main to the unsteadiness of that part of the fusion innervation which remains after the fusion tendency is suspended. Even the relaxation of the fusion innervation obtained by the prism duction test does not last if the eyes are moved from the primary into any secondary direction. Then, as a rule, the deviation decreases slightly; sometimes a vertical component which was not present in the primary direction of gaze will associate with the horizontal one or will disappear or change together with the latter. Not infrequently the amount of the change differs in tests repeated under like conditions. At one time, for example, an esophoria may increase a little in dextroversion; the next time it may decrease or remain unchanged—a behavior different from the physiologic change of horizontal deviations which elevation and depression of the visual plane involve.

The atypical variations of the angle of deviation do not follow any certain rule, and their interpretation may therefore be misleading. Heterophoria is understood as a latent concomitant squint that is made manifest by the suspension of fusion. Concomitant deviations are presumed to be unaltered if the visual lines move only in the horizontal direction. This doctrine must not be taken literally. I do not recall any case of true heterophoria in which I have not found the amount of deviation fluctuating for the primary direction as well as for dextroversion and levoversion when I repeated the same test under like conditions several times in succession or on several days. The same is true for hyperphoria, in which the amount of vertical deviation frequently varies even if it is tested for different vertical directions without changing simultaneously the horizontal direction of gaze. Therefore, I wish to emphasize once more that in cases of heterophoria a test performed but once may lead to wrong conclusions as to the nature and the kind of deviation.

So, in the face of reports on cases in which latent deviation of less than 1 prism diopter was supposed to have caused severe subjective symptoms, I cannot help being skeptical, the more so if the author has neglected to state expressly by what methods and how often the hetero-

phoria was examined. The relief from subjective disorders obtained by prisms compensating for a deviation of one-fourth or one-half arc degree cannot be taken as irrefutable evidence of the decisive rôle played by slight heterophoria as the origin of the subjective trouble. I have seen a number of patients wearing weak prisms prescribed elsewhere who were convinced of the immense service the prisms had rendered in removing severe symptoms. In each case the patient assured me that his symptoms returned as soon as he took off his glasses. Pretending to examine the glasses while the attention of the patient, who had consulted me for a slight conjunctivitis or some other insignificant matter, was diverted by an assistant or a nurse, I rotated the prisms so that they increased the heterophoria instead of correcting it. When I asked the patient on his next visit whether he was still satisfied with his glasses, he replied in the affirmative. On the other hand, I have met with numerous patients who have been given a dozen or more pairs of prism spectacles within a few months, since the prisms that were satisfactory at first after a while became inadequate. The patients would have been saved considerable expense if the amount of heterophoria had been determined accurately in the first examination by adequate and repeated tests. That prisms render the best of service and are even indispensable in many cases of heterophoria is a matter of course. But though I am willing to acknowledge that a comparatively slight amount of hyperphoria may be the cause of serious symptoms, since I have made such trouble disappear promptly with vertical prisms, I should not prescribe prisms of less than 1 degree for both eyes. If the amount of hyperphoria is really not higher than 1 degree I should look for another possible cause of the symptoms and failing to find any I should prescribe prisms of one-half degree only if the amplitude of fusion in the vertical direction was practically zero and an improvement by orthoptic training could not be obtained. But before giving prisms in any case of heterophoria one must make sure that the prisms will correct the deviation not only in a part but in the whole field of fixation.

If heterophoria has remained after paresis of one of the ocular muscles, the patient, having a large fusional amplitude, may be able to overcome the disturbed muscular imbalance even in that part of the field of fixation where the action of the paretic muscle is required. Tests for heterophoria (breaking fusion) will display the paretic character of the deviation only if it is examined while the patient's gaze is directed first to the center and then to the peripheral parts of the field of fixation. If the measurement is confined to the primary direction of gaze the prism correction may be adequate for that direction but inadequate for the secondary directions of gaze, being too weak for one part and too strong for the other part of the field of fixation. Heterophoria of paretic origin with the aforementioned characteristics

does not occur frequently, but it is important with respect to the question of correction.

At the last meeting of the Section on Ophthalmology of the American Medical Association, Dr. J. S. Friedenwald read a paper on the diagnosis and treatment of anisophoria, which he defined as that form of heterophoria in which the degree of muscular imbalance of the eyes varies with the direction of gaze. He separated anisophoria into two types: The first type is heterophoria of paretic origin; the second type is produced by the correction of anisometropia with lenses with different peripheral prismatic effects for the two eyes. Physiologic anisophoria is understood by Friedenwald to be due to variations in heterophoria produced by physiologic factors, for instance, in horizontal deviations by elevation and depression of the visual plane. Slight variations in hyperphoria, frequently found in oblique directions of gaze, seem to depend on a slight inequality of the function of the vertical rectus and the oblique muscles. Though admitting that physiologic anisophoria "like other forms of heterophoria is subject to some variations in amount," Friedenwald said that anisophoria "is a fairly stable element of the ocular movements." From his experience he stated that he considered anisophoria to be present if the patient referred his discomfort to one eye only, complaining especially of pain, lacrimation and photophobia. As to his statement that the least amount of anisophoria which he found to cause symptoms had amounted to 1 prism degree, I must confess that I am skeptical, in view of the aforementioned difficulties in obtaining complete relaxation of the fusion innervation and not knowing how often or by what method the patients had been examined.

I shall not enter into the particulars of Friedenwald's interesting paper. I should like to discuss only the problem of an optical correction of anisophoria, since it is intimately connected with another interesting anomaly, called aniseikonia, and with its correction. An accurate optical correction of anisophoria could be obtained, as Friedenwald stated, by a meniscus lens of no dioptric power, showing a peripheral prismatic action which increases regularly with the eccentricity of the gaze, with the thickness of the lens and with the depth of the meniscus. Such lenses have been developed to correct aniseikonia and were described in 1932 by Ames, Gliddon and Ogle.[4]

4. Ames, A., Jr.; Gliddon, G. H., and Ogle, K. N.: Size and Shape of Ocular Images: I. Methods of Determination and Physiologic Significance, Arch. Ophth. **7**:576 (April) 1932. Ames, A., Jr., and Ogle, K. N.: III. Visual Sensitivity to Differences in the Relative Size of the Ocular Images of the Two Eyes, ibid. **7**:904 (June) 1932. Ames, A., Jr.; Gliddon, G. H., and Ogle, K. N.: III. Lenses for Changing the Size and Shape of Dioptric Images, Ann. Distinguished Serv. Found. Optom. **1**:67, 1932. Ames, A., Jr.; Ogle, K. N., and Gliddon, G. H.: Corresponding Retinal Points, the Horopter and Size and Shape of Ocular Images, J. Optic. Soc. America **22**:538, 1932.

Aniseikonia means inequality in the relative size and shape of the ocular images as they are produced in the brain. The inequality may be caused by differences in the dioptric systems of the two eyes or by a different distribution or size, respectively, of the retinal elements. For the investigation of aniseikonia, its measurement and its correction ophthalmologists are indebted to Ames and his collaborators. Power lenses cannot be used for equalizing the two different images, since they change not only the sizes of the retinal images but also the refraction, a fact resulting in the development of a special meniscus form of zero power lenses. These are called iseikonic lenses and produce either an overall or a meridional magnification. To any iseikonic lens of zero power can be added a power lens, as demanded in cases of ametropia combined with aniseikonia. But of course one must take into consideration that the power lens also has magnification, the amount depending on its power, form and distance from the eye. It is self-evident that iseikonic lenses given to patients with equal ocular images in order to correct anisophoria will produce aniseikonia. On the other hand, if a patient has aniseikonia but no anisophoria the lenses correcting the aniseikonia may produce anisophoria. Referring to patients who have been suffering only from anisophoria and in whom the correcting lenses have produced aniseikonia, Friedenwald pointed to the clinical results which made him express the belief that "such a change may make for greater comfort." But he did not say whether and how he had measured the patients for aniseikonia before and after the correction for anisophoria.[5] A patient with anisophoria may have aniseikonia also, so that the lenses correcting the former anomaly may correct the latter as well. Further, there are many patients with a considerable degree of aniseikonia who make no complaint at all, just as there are many patients with a comparatively high degree of heterophoria without subjective symptoms. Patients are commonly seen who have a considerable degree of anisometropia and who wear the equalizing glasses without any discomfort, in spite of the aniseikonia involved by the correcting lenses. Finally, reference should be made to the statement of Ames and his collaborators that aniseikonia in a great percentage of subjects is different from the aniseikonia induced by the lenses that correct an anisometropia.

CONCLUSIONS

It is impossible to discuss exhaustively the numerous problems that have merely been touched on here. Before one can accept anisophoria as a well determined anomaly that may be segregated from heterophoria

5. In his paper (Diagnosis and Treatment of Anisophoria, Arch. Ophth. **15**:296 [Feb.] 1936) Friedenwald also admitted that "a final decision as to the relative importance of anisophoria and aniseikonia in the production of symptoms can be reached only by making both types of measurement and alternate types of correction in a large group of cases."

by definite and unequivocal signs and symptoms, a number of problems must be solved by means of ample material and careful examinations, taking into account and eliminating as far as possible the numerous contingencies of mistakes and misinterpretations. My reflections may be summarized as follows:

The subjective symptoms reported as characteristics of heterophoria, anisophoria and aniseikonia resemble each other to a high degree, so that they cannot be utilized for a differential diagnosis.

In attempting to separate anisophoria from ordinary heterophoria, the main difficulties to be overcome are, first, the resistance of the compensating fusion innervation and, second, the unsteadiness of the latent deviation even after an almost complete relaxation of the fusion innervation by means of a duction power test. A greater or smaller fraction of that innervation frequently returns even if fusion stimuli are excluded in the examination following the duction power test. That fraction of innervation is not constant but varies in an incalculable way, so that the deviation may appear now as a slight increase and now as a decrease and may induce misinterpretation unless repetitions of the test disclose the unsteadiness of the deviation.

Many patients suffering from disorders considered as symptoms of heterophoria as well as of aniseikonia cannot be helped by careful treatment for heterophoria, i. e., by prism corrections, by exercises or by operation. Only after the aniseikonia has been found and corrected do these patients obtain relief so that they are able to resume their work. I myself have seen and examined some of them before and after the aniseikonia was corrected; I found the amount of heterophoria to be almost unaltered by the iseikonic lenses. This proved, first, that not heterophoria but aniseikonia had caused the trouble and, secondly, that the influence of iseikonic lenses on heterophoria is not as great as could be expected from a theoretical standpoint, considering the peripheral prismatic action of such glasses.

To test for aniseikonia one cannot dispense with the equipment designed by Ames or with a good deal of technical training. Otherwise one is not warranted in judging either the theoretical or the practical significance of the anomaly. Observations on patients with aniseikonia whom I have had the opportunity to examine personally have compelled me to abandon my skepticism as to the clinical importance of aniseikonia, in view of the fact that severe subjective disorders based on objective signs of muscular anomalies that had resisted other therapeutic procedures could be removed by finding and correcting the aniseikonia as the main cause of the trouble.

At a meeting of the Section of Ophthalmology of the New York Academy of Medicine a year ago I demonstrated a patient as presenting

a most striking example of horror fusionis combined with a paretic weakness of almost all the muscles of the right eye.[6] Since the deviation, composed of a lateral, a vertical and a rotary component, varied with the slightest change of the direction of gaze, the condition might have been considered as belonging to Friedenwald's type of anisophoria had not the apparently total deficiency of fusion presented a particular feature that has no relation to anisophoria. Anisophoria for a certain direction of gaze can be corrected by prisms, while anisophoria for different directions can be corrected by adequate lenses of the iseikonic type. Such lenses helped my patient obtain binocular single vision in the whole field of fixation. But this good result had nothing to do with the peripheral prismatic action of the lenses, because their prismatic action was unable to correct the extraordinary variations of the deviation in the different parts of the field of fixation. The prismatic action might have influenced the horizontal deviation component in levoversion and dextroversion or the vertical component in vertical movements, but since in that case a change in the horizontal as well as in the vertical and the rotary component was produced by a purely horizontal as well as by a purely vertical movement of the eyes, the prismatic action of the lenses could not have produced the simultaneous correction of the three deviation components. The most satisfactory result in that case can be explained only by assuming that the equalization of the ocular images removed the only or the main hindrance to the action of the fusion apparatus so that the latter was able to overcome the complicated deviation.

A good many problems remain to be investigated in order to clear up the relation between aniseikonia, anisophoria and heterophoria. The results will help to overcome the difficulties encountered in numerous cases in which there are puzzling discrepancies between severe subjective disorders and apparently insignificant objective findings.

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6. Bielschowsky, A.: Congenital and Acquired Deficiencies of Fusion, Am. J. Ophth. **18**:925 (Oct.) 1935.

HOMONYMOUS HEMIANOPIA

PRACTICAL POINTS IN INTERPRETATION, WITH REPORT OF FORTY-NINE CASES IN WHICH THE LESION IN THE BRAIN WAS VERIFIED

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Since Jackson and Gowers [1] in 1874 called attention to the importance of perimetry in neurologic examinations, it has been known that changes in the visual fields may supply important information as to the general and, at times, the specific location of a lesion of the brain. For example, bitemporal hemianopia, when present, definitely places the lesion at the optic chiasm. When homonymous hemianopia exists the lesion is definitely lateralized on the opposite side, but it may be located at any site along the visual pathway between and including the visual center in the occipital lobe and the chiasm.

In my work at the Neurological Institute of New York sectoral and other incomplete homonymous defects of the visual fields have often seemed to me to have definite localizing significance, and when other symptoms have been correlated with the homonymous changes in the fields the localization of expanding lesions of the brain has been greatly aided. I have had the privilege at the institute of observing a large number of patients with intracranial lesions. In 143 of the cases in which I made perimetric examination there was homonymous hemianopia. The lesion in the brain has been demonstrated at operation or autopsy in 49 of these cases. The patients had been admitted to various services, and those receiving surgical treatment underwent operation in the service of Dr. Charles A. Elsberg.

The study reported in this paper was made with two objects in view: (1) to determine to what extent homonymous hemianopia when correlated with other symptoms of tumor of the brain could be used to localize the growth more specifically and (2) to establish whether the various grades of homonymous hemianopia, such as quadrantic and sectoral defects, could be shown to have specific localizing values.

In this series an effort has been made to group the lesions on the basis of the lobes involved. In classifying lesions of the brain it is to be remembered, however, that the lobes of the brain are continuous structures and that the lesion may be on the anatomic borderline of two

1. Jackson, J. H., and Gowers, W. R.: A Case of Hemiopia, with Hemianesthesia and Hemiplegia, Lancet **2**:306, 1874.

lobes and invade each equally. When the lesion was mainly in one lobe and extended to another, the tumor was classified on the basis of the lobe chiefly involved.

Anatomically, the lesions in the 49 cases were localized as follows: lesion of the frontal and frontoparietal lobe, 5 cases; lesion of the temporal lobe, 20 cases; lesion of the temporal lobe but extending into the adjoining lobes, 8 cases; lesion of the pituitary gland, 1 case; porencephaly and degeneration of the parietotemporal lobes, 3 cases; angioma of the right side of the tegmentum, 1 case, and lesion of the occipital lobe or primarily originating in the occipital lobe and invading the adjoining lobes, 11 cases.

TUMOR OF THE FRONTAL LOBE

In this series there were 5 cases in which a tumor of the frontal lobe had produced homonymous hemianopia by pressure on the optic tract. In 4 there was complete, and in 1 incomplete, homonymous hemianopia. One patient had paralysis of the opposite side of the face due to the tumor; 1 had no papilledema, and 2 had bilateral papilledema; 1 showed the syndrome of atrophy of the optic nerve on the ipsilateral and papilledema on the contralateral side, and in 1 there was optic atrophy on the contralateral and papilledema on the ipsilateral side. Generalized convulsive seizures had been present in 2 cases, and paresis of one or both contralateral extremities, in 3 cases. In only 1 instance had there been hallucinations of smell, and in this instance the tumor had invaded the temporal lobe.

Symptoms of tumor of the frontal lobe vary greatly, depending on whether the growth involves the right or the left lobe and whether it is dorsal or ventral, anterior or posterior.

A tumor may exist in the frontal lobe for a long time with no localizing symptoms. As this region is probably the seat of intelligence, changes in the mental state and in personality are likely to occur. The anterosuperior part of the lobe may be involved, with only a few general symptoms becoming manifest. When the growth causes pressure on the motor area, twitchings of the contralateral extremities occur, followed by convulsive seizures and loss of consciousness as the pressure increases. Finally, complete hemiplegia may follow if the tumor invades the motor area. A tumor at the base of the frontal lobe in the early stage may produce symptoms of retrobulbar neuritis, accompanied by a central or paracentral scotoma in the ipsilateral visual field. By pressure on the olfactory bulb, neoplasms in that location often alter the sense of smell on the same side. Exophthalmos is common in cases of meningioma of this lobe.

In my experience the syndrome of ipsilateral optic atrophy with contralateral papilledema is not uncommon in cases of tumor of the frontal lobe. There has been difference of opinion among observers as to the prevalence and importance of this syndrome. Kubitschek [2] in the study of 22 cases of tumor of the frontal lobe had no case in which unilateral atrophy of the optic nerve was accompanied by papilledema in the other eye. Sachs,[3] in a report of 25 cases of verified tumor of the frontal lobe, had a similar experience. Kennedy, Holmes and Lillie have noted the not infrequent occurrence of this syndrome. It is believed that the explanation for the difference in the observations of these authors lies in the location of the tumor. It is probable that only a tumor at the base of the lobe produces the characteristic changes in the optic nerve heads. When the syndrome is present, however, it has definite localizing significance.

A tumor affecting the postero-inferior portion of the left frontal lobe in right-handed persons is likely to be accompanied by motor aphasia. A neoplasm that by pressure irritates the middle frontal gyrus causes conjugate deviation of the eyes to the ipsilateral side; a tumor that destroys the center by pressure or invasion prevents the conjugate movement of the eyes to the ipsilateral side.

A tumor in the postero-inferior part of the lobe is more likely to bring about changes in the visual fields. This area is in close relation to the optic tract, chiasm and nerve. A neoplasm in this location may at the inception produce central scotoma on the same side by subjecting the optic nerve to the influence of toxins thrown off by the tumor or to pressure. Growth of the neoplasm may cause by pressure complete atrophy of the optic nerve with total loss of vision of the eye on that side. It is not uncommon for a basal tumor of the frontal lobe to extend posteriorly, causing destructive pressure on the ipsilateral optic nerve and the chiasm, producing complete blindness in the ipsilateral visual field and temporal blindness in the contralateral field. When the tumor is well back in the portion of the frontal lobe bordering on the frontotemporal region, the optic nerve and chiasm may escape injury, and pressure may be produced on the ipsilateral optic tract, thus causing contralateral hemianopia. In the cases of tumor of the frontal lobe reported in this series, the growth was in this location.

In many cases of tumor of the frontal lobe no defects of the visual fields are shown. Sachs [3] found a single instance of central scotoma

2. Kubitschek, P. E.: The Symptomatology of Tumors of the Frontal Lobe, Based on a Series of Twenty-Two Cases, Arch. Neurol. & Psychiat. **20**:559 (Sept.) 1928.

3. Sachs, Ernest: Symptomatology of a Group of Frontal Lobe Lesions, Brain **50**:474, 1927.

and 5 cases of partial homonymous hemianopia in 25 cases of verified tumor of the frontal lobe. He expressed the belief that the homonymous changes in the field were due to invasion of the temporal lobe. Voris, Adson and Moersch [4] found defects of the visual field in 32 per cent of a series of 314 cases of verified tumor of the frontal lobe. They called attention to an important feature of perimetric study in cases of brain tumor, viz., that many patients cannot cooperate sufficiently well so that satisfactory plottings of the fields are yielded. They did not state the character of the changes found in the visual fields in their cases.

Comment.—Defects in the visual fields are common in cases of tumor at the base of the frontal lobe. When the pressure is not directly exerted on the visual structures but on areas adjacent to them, the change is likely to consist of a central scotoma in the ipsilateral field. When the pressure is produced on the optic nerve and tract near the chiasm, there may be total loss of the ipsilateral field and temporal loss of the contralateral field. Pressure confined to the optic tract produces contralateral homonymous hemianopia, which is likely to be complete in the proportion of 4:1 of the cases.

When a group of symptoms indicates the presence of tumor of the frontal lobe and there is accompanying homonymous hemianopia, the postero-inferior part of the contralateral lobe is the site of the lesion.

TUMOR OF THE TEMPORAL LOBE

In this series the temporal lobe was involved in 28 cases. In 20 instances the lesion was practically limited to the temporal lobe, and in 8 the contiguous lobes were invaded. Weakness of the face was present in 21 of the 28 cases, contralaterally in 19 and ipsilaterally in 2. Complete homonymous hemianopia was present in 7 cases, and incomplete, in 21 cases. In 3 of the cases of incomplete hemianopia the defects were quadrantic, and in 7, crescentic. Papilledema was present in 21 instances, general convulsive seizures in 10 and paresis of one or both of the contralateral extremities in 12.

The following symptoms are common in cases of tumor of the temporal lobe: epileptiform convulsions, dreamy states and subjective sensations of smell or taste, with or without involuntary movements of mastication. On the contralateral side there may be transient weakness of the lower part of the face, diminution or absence of abdominal reflexes and, less often, transient paresis of the arm and leg. No word forgetfulness or speech defect occurs in a right-handed person when the right lobe alone is involved.

4. Voris, H. C.; Adson, A. W., and Moersch, F. P.: Tumors of the Frontal Lobe, J. A. M. A. **104**:93 (Jan. 12) 1935.

When the left temporal lobe is the site of the lesion, because of the presence of speech centers in that lobe, the following symptoms are added to the complex: difficulty in naming objects, word forgetfulness in conversation and later the use of inapposite words and phrases, with instant recognition of, but inability to prevent, the mistake.

There may be transitory but recurring ptosis and pupillary changes, the commonest of these being ipsilateral myosis. In addition, the pupil may become permanently dilated as the tumor grows larger.

In this series weakness of the opposite side of the face was found in 67 per cent of the cases of tumor of the temporal lobe. Kolodny [5] observed weakness of the lower part of the opposite side of the face in 66 per cent of 38 cases of verified tumor of the temporal lobe.

As the optic radiations pass through the full length of this lobe and are subject to pressure or invasion, changes in the visual fields are prevalent. Depending on the part of the radiations involved, the blind areas vary in extent and location and may have localizing significance. Homonymous hemianopic defects have been more frequently found in cases of tumor of the temporal lobe than in those of any other part of the brain, in my experience. This may be due to the greater prevalence of tumor in the temporal lobes.

In the present series of 28 cases of expanding lesion of the temporal lobe, homonymous hemianopia was incomplete in 21 instances. The incomplete defects seem to be significant of invasion of the temporal lobe. These findings support the views of Cushing and Heuer.[6] They stated that partial defects in the fields were especially characteristic of involvement of the optic radiations of the temporal lobe. They expressed the belief that a lesion situated in the posterior part of the lobe is less likely to produce partial defects in the fields or to be detected than one that involves the optic radiations as such in the anterior segment of the lobe. They concluded that perimetry gives information of paramount importance in cases of tumor of the temporal lobe.

Kolodny,[5] in a series of 38 cases of tumor of the temporal lobe, found that perimetry was of value in diagnosis in only 7 instances, or in 18.5 per cent of the cases. This caused him to doubt the validity of the statement of Cushing and Heuer that "the perimeter as a diagnostic aid in temporal lobe tumors is possibly the most important agent of all." Kolodny stated that, as the optic radiations pass through the temporal lobe, changes in the visual fields would be expected in most

5. Kolodny, Anatole: The Symptomatology of Tumors of the Temporal Lobe, Brain **51**:385, 1928.

6. Cushing, Harvey, and Heuer, G. J.: (*a*) Distortions of the Visual Fields in Cases of Brain Tumor, Bull. Johns Hopkins Hosp. **22**:190, 1911; (*b*) Distortions of the Visual Fields in Cases of Brain Tumor: The Field Defects Produced by Temporal Lobe Lesions, Tr. Am. Neurol. A., 1921, p. 374.

cases of tumor of the temporal lobe but that certain anatomic considerations suggest an explanation of the actual clinical facts. The optic radiations in part pass around the walls of the inferior and posterior horns of the lateral ventricle. He expressed the belief that the fibers of the visual pathways in that location are less vulnerable than elsewhere. As the tissues in the neighborhood of the ventricle are more elastic and yielding, the wall of the ventricle gives way before the pressure. He expressed the belief that the fibers in the posterior half of the temporal lobe are more compact and lie in tissues which are more resistant to pressure. His opinion was, therefore, that a tumor in the posterior half of the temporal lobe is more likely to cause homonymous hemianopia than one in the anterior part.

Comment.—A large part of the temporal lobe is in the so-called silent area of the brain, and a neoplasm in this region may reach a large size and produce no localizing signs.

The speech centers are confined to the left temporal lobe (in right-handed persons) and may aid in localizing a tumor of that lobe, but they offer no help when the growth is in the right lobe. The centers for smell are located in the temporal lobes, and when they are affected they may produce hallucinations of smell, but such hallucinations are not frequently encountered. The centers for hearing, which are located in these lobes, have bilateral cortical representation and usually present no localizing symptoms. The optic radiations, occupying a considerable portion of each lobe, are expected to, and do, supply the most important localizing evidence when they are implicated by the resulting homonymous hemianopic changes in the visual fields, the incomplete defects being particularly characteristic. Visual hallucinations, consisting of scenes and panoramas due to irritation of the optic radiation, are diagnostic of tumor of the temporal lobe.

LESION OF THE OCCIPITAL LOBE

Eleven cases of lesion of the occipital lobe occurred in the series; 10 of the lesions were tumors and 1 a bullet wound. In 4 cases the tumor was confined to the occipital lobe; in 5 it extended to the parietal lobe, and in 1, to both the parietal and the temporal lobe. In 5 of the cases of tumor there was bilateral papilledema; in 3 weakness of the opposite side of the face was shown, and in 6 complete and in 3 incomplete homonymous hemianopia. Mild convulsive seizures occurred in 1 case and major seizures in another. There was weakness of one or more extremities in 3 cases.

A tumor confined to the occipital lobe causes few symptoms. Pressure on the cerebellum may bring about ataxia and instability of station. Mind blindness may occur. Homonymous hemianopia follows impli

cation of the visual center. In the beginning the field defect may be quadrantic, progressing to complete half-blindness as the growth increases. Rarely, a tumor in the midline affects both calcarine areas, causing bilateral homonymous hemianopia. Visual hallucinations, consisting of flashes of light and spectroscopic luminosities, are significant of invasion of the occipital lobe.

Tumor of the occipital lobe is less common than tumor of the temporal or frontal lobe. Bramwell [7] reported that he had observed 122 cases of tumor of the brain, in 40 of which autopsy had been performed. In only 2 of the 40 cases was the occipital lobe involved. Dowman and Smith [8] observed tumor in the occipital lobe in 2 of a series of 100 cases of verified tumor of the brain. Cairns [9] noted tumor of the occipital lobe in 9 of 162 reported cases of verified cerebral neoplasm. Horrax and Putnam [10] observed tumor of the occipital lobe in 40 of a series of 1,881 cases of verified tumor of the brain at the Peter Bent Brigham Hospital.

Allen,[11] in a study of 40 cases of tumor of the occipital lobe, found that the visual fields were normal in 5 per cent; in 32.5 per cent there was contralateral homonymous hemianopia, extending to the point of fixation; in 22.5 per cent there was contralateral homonymous hemianopia, extending to the midline above and below the field for central vision but leaving the latter area more or less unaffected; in 7.5 per cent there was a contralateral homonymous crescentic defect, and in 12.5 per cent, a contralateral homonymous quadrantic defect; in 20 per cent the patient's condition prevented the taking of satisfactory fields. Allen stated: "The defects in the visual fields found in association with tumor of the occipital lobe were thus shown to vary considerably with the position of the tumor and the degree to which it affected the fibers of the optic radiation."

Traquair [12] expressed the belief that hemianopia as an isolated symptom is due so rarely to a lesion of the optic radiation and so fre-

7. Bramwell, B.: On the Localization of Intra-Cranial Tumors, Brain **22**: 1, 1899.

8. Dowman, C. E., and Smith, W. A.: Intracranial Tumors: A Review of One Hundred Cases, Arch. Neurol. & Psychiat. **20**:1312 (Dec.) 1928.

9. Cairns, H.: A Study of Intracranial Surgery, Medical Research Council, Special Report Series, no. 125, London, His Majesty's Stationery Office, 1929.

10. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumour: The Field Defects and Hallucinations Produced by Tumours of the Occipital Lobe, Brain **55**:499, 1932.

11. Allen, I. M.: A Clinical Study of Tumors Involving the Occipital Lobe, Brain **53**:194, 1933.

12. Traquair, H. M.: Fields of Vision in Intra-Cranial Lesions, Brit. M. J. **2**:229, 1933.

quently to a lesion of the occipital cortex that the lesion may be supposed to be in the latter position unless there is evidence, such as a wound, to indicate that the optic radiation has been injured.

Frazier and Waggoner [13] concluded that the association of hemianopia and visual aphasia points to a tumor of the occipital lobe.

Comment.—Homonymous hemianopia is seen much more frequently in cases of tumor of the temporal lobe than in those of the occipital lobe because tumor of the occipital lobe is relatively much less common.

Homonymous hemianopia is more likely to be complete than incomplete when the occipital lobe is the site of the growth.

As far as is known, the only function of the occipital lobe is visual, and the localizing signs of a lesion of this lobe are expected to consist of homonymous defects of the visual fields and possibly of visual hallucinations and visual aphasia.

VARIOUS TYPES OF HOMONYMOUS DEFECTS OF THE VISUAL FIELDS

Do incomplete homonymous defects of the fields have specific localizing value?

A great deal has been written about the specific diagnostic importance of what Weyman [14] called "quadrisectoral homonymous hemianopia" and Cushing and Heuer [6] later termed "quadrantopsia." The reports of numerous recent authors have left the impression that quadrantic homonymous defects of the visual fields signify tumor of the temporal lobe.

There has been no unanimity of opinion in the interpretation of changes in cases of tumor of the brain. Observers have sometimes attributed specificity of a particular symptom to a given region, only to be contradicted by others, who assign the same symptom to totally different zones. Facial paresis is an example. Sachs [3] considered it to be a constant representative of tumor of the frontal lobe. However, Allen [11] found it in 57.5 per cent of 40 cases of verified tumor of the occipital lobe. A similar situation exists in homonymous sectoral defects of the visual fields. To many observers such defects have appeared to be characteristic of tumor of the temporal lobe. But Allen found the sign to be significant of tumor of the occipital lobe in 12.5 per cent of the 40 verified cases in his series. On the other hand, he found a homonymous crescentic field defect in 7.5 per cent of the cases, while no crescentic alterations occurred in the 10 cases of tumor of the

13. Frazier, C. H., and Waggoner, Raymond: Tumors of the Occipital Lobe, Arch. Neurol & Psychiat. **22**:1096 (Nov.) 1929.

14. Weyman, M. P.: A Case of Quadrisectoral Homonymous Hemianopia, Am. J. Ophth. **11**:289, 1894.

occipital lobe in the series reported in this paper, but in 6 of the 28 cases of involvement of the temporal lobe such a defect was shown.

Numerous cases have been reported in which quadrantic defects have been caused by lesions of the various structures that constitute the visual pathways.

Weithe [15] reported the case of a man who had had a stroke, after which superior homonymous quadrantanopia was noted. At autopsy numerous hemorrhages were observed in the brain, one of which was in the optic thalamus of the opposite side.

Hun [16] described the case of a man, aged 57, who had left inferior homonymous quadrantanopia. At autopsy complete atrophy of a convolution on the medial surface of the right occipital lobe was noted. The atrophy was strictly limited to the lower half of the cuneus.

Weyman [14] contributed the report of a case of a "dancing professor," who was paralyzed and had quadrisectoral homonymous hemianopia. The lesion in the brain was not verified.

Henschen [17] reported a case in which inferior homonymous quadrantanopia was produced by a hemorrhagic cyst in the posterior part of the pulvinar, which had also destroyed the dorsal half of the external geniculate body of the opposite side. The optic tract and radiations were intact.

Harris [18] reported the case of a man, aged 43, who was seized suddenly by left hemiplegia, partial left hemianesthesia and complete left inferior homonymous quadrantanopia. At autopsy softening was observed in the right internal capsule.

Schiff-Wertheimer [19] described a case of Masson's in which superior quadrantic homonymous hemianopia was observed to be due to a lesion of the optic thalamus of the opposite side in a man of 60. The author included a case of her own, that of a man, aged 60, who had right superior homonymous quadrantanopia. At autopsy a lesion of the left cuneus was noted, which was limited to the inferior lip of the calcarine fissure.

Cushing and Heuer [6] were the first to stress the importance of quadrisectoral homonymous changes in the fields in cases of tumor of

15. Weithe, Theodore: A Case of Amaurosis Followed by Homonymous Superior Hemianopia, Arch. Ophth. **13**:301, 1884.

16. Hun, Henry: A Clinical Study of Cerebral Localization, Am. J. M. Sc. **93**:141, 1887.

17. Henschen, S. E.: Klinische und anatomische Beiträge zur Pathologie des Gehirns, Uppsala, Almqvist & Wiksells, 1890-1922, vols. 1-7.

18. Harris, W.: Hemianopia with Especial Reference to Transient Varieties, Brain **20**:364, 1897.

19. Schiff-Wertheimer, S.: Les syndromes hémianopsiques dans le ramoullissement cérébral, Thèse de Paris, no. 66, 1926.

the temporal lobe. They expressed the belief that enough attention had not been paid to Meyer's discovery of the course of the ventral fibers of the optic radiations forward and downward around the temporal horn of the lateral ventricle. In their classic paper,[6b] they reported cases in which a correct diagnosis of neoplasm in the anterior tip of the temporal lobe had been made on the basis of such sectoral defects, which they stated to be due to the invasion of Meyer's loop.

Lillie,[20] Sheldon and Lillie,[21] Kravitz,[22] Horrax and Putnam [10] and Cave [23] supported the view of Cushing and Heuer by reporting cases of tumor of the temporal lobe associated with homonymous quadrisectoral field defects.

However, as eminent an authority as Traquair [12] expressed a doubt whether Meyer's loop is a part of the connection between the geniculate body and the occipital lobe.

Allen [11] expressed the belief that quadrantic defects, usually described as characteristic of tumor of the temporal lobe, are equally frequent in cases of small tumor involving the calcarine fissure and of large tumor invading the optic radiation from the inner aspect, or from below when situated in the anterior part of the temporal lobe.

In this series complete homonymous hemianopia was shown in 4 of the cases of tumor of the frontal lobe and incomplete homonymous hemianopia, in 1 case.

In 7 of the cases of lesions of the temporal lobe complete homonymous hemianopia was present, and in 21, incomplete.

Of the 21 cases of incomplete hemianopia, quadrisectoral defects occurred in 4 and crescentic defects in 6.

Of the cases of tumors of the occipital lobe, complete homonymous hemianopia occurred in 7 and incomplete hemianopia in 4 cases. In 1 case of incomplete hemianopia the defect was quadrantic, but in the other 3 instances it was of the ordinary variety.

In the remaining cases the lesion was so extensive as to include most of the optic radiations.

Comment.—Homonymous sectoral field defects indicate a lesion in the visual system posterior to the chiasm, but they do not locate the lesion in any specific area.

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20. Lillie, W. I.: Ocular Phenomena Produced by Temporal Lobe Tumor, J. A. M. A. **85**:1465 (Nov. 7) 1925.

21. Sheldon, W. D., and Lillie, W. I.: Importance of Visual Fields as an Aid in Localization of Brain Tumors, J. A. M. A. **94**:677 (March 8) 1929.

22. Kravitz, Daniel: The Value of Quadrant Field Defects in the Localization of Temporal Lobe Tumors, Am. J. Ophth. **14**:781, 1931.

23. Cave, H. A.: Temporal Lobe Lesions: Disturbances of the Visual Pathways, California & West. Med. **36**:13, 1922.

Tumor of the frontal lobe rarely causes incomplete homonymous hemianopia. Tumor of the temporal lobe produces incomplete homonymous defects much more frequently than tumor of the occipital lobe.

EVALUATION OF CHANGES IN THE VISUAL FIELDS IN CASES IN WHICH HOMONYMOUS HEMIANOPIA IS PRESENT

There are no homonymous hemianopic changes in the fields which represent involvement of any specific area of the brain.

It is obvious that an incisure into the visual pathway from any direction produces a sectoral field defect which can be detected if the patient is seen early enough in the course of the disease and careful perimetric studies are made. The lesion may be at any site between and including the cortical visual center and the optic chiasm.

A sectoral defect in the lower portion of the field shows that the lesion lies at a level superior to that of the visual pathway; one in the upper part of the field shows that the reverse is true.

Crescentic defects indicate that the lesion is external to the visual pathway and in the same lateral plane.

A permanent homonymous defect is a positive sign of an organic lesion involving the visual system of the opposite side.

A homonymous defect with papilledema definitely indicates the presence of an expanding lesion of the brain.

Homonymous hemianopia accompanied by hemiplegia of the same side and papilledema, but without hemianesthesia, points to a tumor in the posterior part of the frontal lobe. In a right-handed person aphasia may also be a part of the syndrome if the lesion is on the left side.

A homonymous defect accompanied by atrophy of the optic nerve on one side and papilledema on the other suggests a tumor in the postero-inferior part of the lobe contralateral to the field defect. If anosmia is also present, a lesion of the frontal lobe is definitely indicated.

A homonymous defect accompanied by so-called uncinate attacks (hallucinations of smell and taste, sometimes with chewing movements of the mouth and lips and dreamy states) or hallucinations of vision consisting of pictured scenes points to a tumor of the temporal lobe on the side opposite that of the field defect. If aphasia or anomia is also present the temporal lobe is more certainly implicated.

When a homonymous field defect is associated with definite ipsilateral sensory changes and papilledema, tumor of the temporoparietal lobe of the side opposite that showing the sensory signs is to be suspected.

Homonymous hemianopia, hemianesthesia, choreiform or athetoid movements and slight hemiparesis of the same side constitute the most typical syndrome of a lesion of the optic thalamus of the opposite side.

Homonymous hemianopia associated with hemianesthesia, hemiplegia of the same side and papilledema suggests a tumor situated deep in the temporal or the occipital lobe of the opposite side.

Homonymous hemianopia, papilledema and visual hallucinations, consisting of flashes of light of spectroscopic luminosities, indicate tumor of the occipital lobe. Mind blindness is also commonly attributed to a lesion of the occipital lobe.

DIFFERENTIATION OF EXPANDING AND VASCULAR LESIONS OF THE BRAIN

Hemorrhage into, or thrombosis of the blood vessels supplying, the cerebral visual structures produces a sudden loss of vision of which the patient is tragically conscious. When homonymous hemianopia results, the form of the blind area is likely to be irregular, with jagged peaks and depressions. If the patient survives there is commonly some restoration of visual acuity, and the field defect soon becomes stationary. The retinal arteries are usually sclerotic.

In cases of an expanding lesion of the brain, it is common for the patient to be unaware of the loss in the visual fields until he bumps into objects. In a case observed at the Neurological Institute of New York, the patient had not been conscious of any visual defect until he walked into one of the steel pillars of an elevated railway. Generally, persons subconsciously adjust themselves to slowly developing impairment of the visual fields, provided that central vision remains good. In addition, the visual loss is likely to be progressive in cases of tumor of the brain.

CONCLUSIONS

1. Homonymous hemianopia occurring in association with tumor of the brain is incomplete in the following proportion of cases: in tumor of the frontal lobe, 1:4; in cases of tumor of the temporal lobe, 20:7, and in cases of tumor of the occipital lobe, 4:6.

2. It is to be expected that cases of tumor of the temporal lobe with incomplete homonymous hemianopia will predominate, as tumor of the temporal lobe is much more frequently associated with hemianopia than is tumor of the frontal lobes and the incidence is much greater than that of tumor of the occipital lobe.

3. Crescentic defects in the visual fields are not as significant of tumor of the occipital lobe as of that of the temporal lobe.

4. Crescentic defects are seen as frequently as quadrisectoral defects in cases of tumor of the temporal lobe.

5. Central vision in homonymous hemianopia is retained, as a rule, until secondary atrophy of the optic nerve has resulted from concomitant papilledema.

6. A homonymous field defect of the slightest degree accompanied by papilledema is strongly significant of tumor of the brain.

7. Careful perimetric examination at close angulation should be made in every case in which there is any suggestion of the presence of an intracranial lesion.

8. Encephalography or ventriculography is indicated in any case of an obscure neurologic lesion with a progressive hemianopic field defect, particularly if papilledema is present.

9. Homonymous hemianopia does not represent involvement of any specific area of the brain. The lesion may be in the visual system at any site posterior to the optic chiasm.

CONTACT GLASSES, THE "INVISIBLE" SPECTACLES

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The theory of "invisible" eyeglasses, which in 1827 was expounded by Herschel, the English physicist, was again advocated at the end of the eighties of the last century by three German scientists almost simultaneously. All three independently reached the conclusion that contact glasses are of value by working on different problems, the idea of the invisibility of the eyeglasses not being considered.

A. E. Fick, ophthalmologist of Zurich, was studying the problem of correction of so-called irregular astigmatism. One of the principal conditions of clear vision is that the shining, crystal-clear frontal lens of the eyeball, the spherically vaulted cornea, must be perfectly healthy. If the cornea is flattened in one direction, which may be congenital or the result of an operation, the so-called regular astigmatism, originating from the aforementioned causes, can be corrected by cylindric eyeglasses. But the cornea, because of its superficial position, is frequently exposed to injuries as well as to the common phlyctenular, trachomatous and parenchymatous inflammations, which cause it to heal with scarring and resulting irregular deformation. Weak sight resulting from these causes cannot be helped by regularly ground eyeglasses. Sad to say, smoothing of the cornea to a mirror-like spherical surface is an impossibility. Extirpation of the deformed cornea and transplantation of a new one are highly dangerous, since the fate of corneal grafts, which sooner or later lose their transparency, is well known. Fick pondered on the possibility of excluding the cornea at least optically and replacing its refraction of light by a regular spherical surface. For example, if an egg is broken into a glass of water, it appears as if only the yolk of the egg were suspended there, the white of the egg seeming to have disappeared. In like manner the protein material of the cornea of the living eye becomes invisible when under water. With these principles in mind Fick asked the late Professor Abbé, director of the Zeiss factory, in Jena, to construct a thin, precisely ground glass bowl with a spherical surface which could be placed on the domelike surface of the eyeball. If such a glass bowl (*Kontaktbrille*, touching spectacle) is placed behind the eyelids so that the space between the glass and the eyeball becomes filled with lacrimal fluid or is filled up with a similar

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fluid (Fick could not have had at that time any conception of isotony), then the cornea under the layer of fluid between the eyeball and the glass bowl adhering to it disappears optically, and its rôle is taken over by the central part of the accurately ground spherical glass bowl as a new, artificial glass cornea. The eye therefore recovers its power of regular refraction and sharp vision.

The experiments performed by Fick succeeded wonderfully, and his reports were highly optimistic. However, the dissertation published by a second investigator, the young August Müller, medical student at Kiel, was most pessimistic. His thesis contained a discussion of the advantages of the then latest curved lenses (periscopic, meniscus glasses) over the other more simply ground eyeglasses. Today these advantages are well known; through the periphery of meniscus glasses one sees just as clearly as through the center. The price of sharp vision, however, is high if the person who does not see well wears spectacles, because no matter how perfectly the glasses are ground they cause distortion: Cylindrical glasses elongate objects, and concave glasses reduce the size; the stronger the glasses the more easily is dizziness induced. Therefore, a person with a high degree of myopia usually cannot wear the glasses of total correction, and a subject with aphakia who has gained back his sight after an operation for cataract, because strong lenses must be worn, must learn to walk anew and avoid bumping into objects. Many such persons feel their way and stumble to the end. The frame of the spectacles decreases the field of vision and the area of sight. Rain in fall and vapor in winter are great handicaps to any one who wears spectacles. Young persons often cannot take part in sports because they must wear glasses. How much more perfect it would be to place directly on the eyeball a small glass lens which would adhere and move with the eye. August Müller had myopia of — 14.0 diopters. He calculated the measurements of a small glass lens which would correct his sight, and Himmler, an optician of Berlin, succeeded in grinding such a curved, thin, spherical glass lens. Happy as Müller was when he adjusted his lens and experienced himself that his theory was correct, he had to admit after many lengthy experiments that the small "corneal lenses" (*Hornhautlinse*) cannot be worn for a long time, because they irritate the eyes.

It was at this point that a third German investigator, F. A. Müller, glassblower of Wiesbaden, artistic constructor of artificial eyes, began his work, for which he was made an honorary doctor of medicine. Müller had had years of experience in making blown glass prostheses, having engaged in that work ever since Saemisch had sent a wretched patient to him. This 60 year old man was blind in the left eye, and cancer had destroyed both eyelids of the right eye. The cornea of

the unprotected eye had begun to dry out, and symptoms of approaching destruction (keratitis e lagophthalmo) had appeared. Müller was requested to prepare a prosthesis that would replace the missing eyelids and cover and protect the eye but in such a manner that the patient's sight would not suffer. Müller prepared a protective shell comparable to those that he was accustomed to apply on atrophied stumps, an almond-shaped, thin, whitish glass shell with fine blood vessels sketched on the scleral part; but, instead of a painted iris and a black spot for the pupil, he blew a hemisphere of thin, transparent glass in the center. The patient wore this protecting shell for over twenty years! This first patient was followed by many more, some of them requiring lenses not for protection but for improvement of vision. In time Müller learned how to form the shells so that they would lie evenly on the eyeball and how to shape the central transparent glass cornea so that it would improve the vision. Most of the patients were able to wear the shells all day long without any pain, redness, tearing, feeling of a foreign body or photophobia, in other words, without any irritation or discomfort. A few of these patients were presented before medical societies, and it slowly became known that the Müller type of blown shells were wearable, practical ophthalmic prostheses for the protection and improvement of vision.

Why ophthalmologists still did not accept this invention can readily be explained. Many questioned how a sensitive eye can bear the presence of a large piece of glass when a tiny particle of dust causes pain. The timid ones feared the causation of injury or infection and did not dare to shoulder the responsibility. Furthermore, it is understandable that ophthalmologists, who were accustomed to prescribe precisely the type of eyeglasses required according to diopters, were disinclined to experiment at random with glass shells of imperfect dioptric effect which could not be numbered or classified.

For this reason they more gratefully accepted the ground contact glasses, devised by Sulzer, which carried out Fick's theory. After Sulzer's enthusiastic report had been published many attempted to verify his results. However, these controlling experiments failed, just as had those of the young August Müller. Thus in the first quarter of the century the accepted point of view in ophthalmology was that blown shells can be worn in certain cases but that their optical correcting power is limited and that ground glasses, which have an ideal optical corrective effect, can be used only for scientific or diagnostic purposes, not in practice.

It was only too evident that many authors desired to combine the advantages of the haptic (fitting) qualities of the blown type and the optical qualities of the ground type of contact glasses. This, however,

proved to be technically impossible, because a large series of blown shells were all broken during optical grinding.

There was no other way out but to perfect the forms of the ground shells. It was the Zeiss factory, in Jena, which undertook the performing of this task. The margins of round, spherical glass bowls, 0.5 mm. thick and 20 mm. in diameter, were rounded off; the curvature of the inner surface of the scleral part was standardized with the radius of 12 mm.; the inner edge on the boundary between the cornea and sclera was smoothed (Hartinger), and four types of different corneal curvatures were established; two flat and two curved types were brought into circulation. The results were hardly better than those with the Sulzer type of ground shells. Therefore, from 1930 on, according to the proposals of Heine, professor of ophthalmology in Kiel, a variable series of glass bowls were ground with corneal curvatures of from 5 to 11 mm. and scleral curvatures of from 10 to 14 mm., with a gradation of 0.25 mm. Since then experiments have been carried on everywhere with this Zeiss type of ground contact glasses (*Haftglas,* "adhering glass"). The first reports were prompted by a burst of enthusiasm, which was understandable. Patients who had resigned themselves to their fate and those who had not been able to give up hope of recovering their sight, but who had been forced to abandon their careers and had been deprived of many pleasures, felt that a miracle had been performed by the one who first put contact glasses on their eyes. And so it is no wonder that the ophthalmologist, who is not accustomed to seeing a passionate outburst of happiness in his patients, has been absorbed by their trustful enthusiasm and without awaiting the necessary time for the drawing of conclusions from adequate experience, but with unlimited optimism, has offered his opinion of the ground Zeiss glasses, with the optical precision that they offer.

Experience has shown that the more types of ground bowls at hand, the easier it is to find a bearable one (Much); however, in spite of the countless number of combinations of curvatures, sizes (Sattler) and forms (Erggelet) of these glasses, their use is only in rare cases satisfactory. In the ophthalmologic clinic of the University of Kiel, where there is a series of over three hundred different types of ground contact glasses (valued at about $10,000) only a very small percentage of those who desire contact glasses can be satisfied.

The cause of the difficulties of fitting these glasses can be understood if the mode of fitting them is studied. The inner surface of the watery adhering contact glass which bears the pressure of the eyelids must fit the eyeball so that it neither injures the sensitive tissues nor interferes with the circulation. The ground spherical glasses do not and cannot satisfy these requirements, because the eyeball is not a sphere. Models

prepared by making a cast of the living eye (August Müller, Csapody and others) show absolutely that on the surface of the eyeball only the center of the cornea approaches the surface of a sphere and that it flattens out toward the limbus, while on the boundary of the sclera it continues evenly to the gradually flattening curve of the protective bulbar conjunctiva. It is noted that besides this the curvature of the scleral conjunctiva usually changes in every meridian. It is evident that the supposition is proper that a shell which will exactly fit the surface of the eye according to these requirements will far excel in haptics, i. e., fitting and comfort, the ground spherical contact glasses, which are geometric forms that do not follow the anatomicophysiologic conditions of the eye; and because of their spherical form their edges cut into the soft tissues of the nonspherical eyeball instead of fitting its surface uniformly.

This point could not be proved until it was possible to make an extensive, exact cast of the living eye and through this means to construct an accurately fitting glass shell. In 1932 I succeeded in finding a material, Poller's [1] negocoll, which was suitable for the purpose of making perfect casts and devised a method of making the casts. The principle of the method is that an approximately fitting glass shell is filled with a soft paste of boiled negocoll, cooled to room temperature and placed behind the eyelids on the eyeball, which has been anesthetized with cocaine. Twenty or thirty seconds later the shell with the adhering elastic film negocoll, which in the interim has jellied, is removed. The rigid glass shell prevents deformation of the film of negocoll, representing the negative cast of the eyeball, so that with a suitable substance, for example, Poller's hominit, an undistorted, exact positive mold of a large area of the anterior surface of the eyeball can be made. The procedure is no more disagreeable than the methods used in dentistry.

To prepare accurately fitting glass shells to match the molds offered greater difficulties. At various institutes of glass technic painstaking but unsuccessful attempts were made. Finally the bacteriologic laboratory of the Ophthalmologic Clinic No. 1, entirely unequipped for technical experiments, was the birthplace of the new technical method that makes possible the preparation of a thin glass shell with an inner surface that exactly corresponds to the outer surface of the mold.

By means of this new method I soon prepared several glass shells that precisely corresponded to the molds taken from the eyes; but the patients could not wear them. The eyelids pressed on the shell, the soft conjunctiva receded because of the pressure and the shell

1. Poller, Alphons: Das Pollersch Verfahren zum Abformen an Lebenden und Tote, sowie an Gegenständen, Berlin, Urban & Schwarzenberg, 1931.

wavered about on the hard, taut but extremely sensitive cornea. When the form of the shell was modified new difficulties arose, and only with the assistance of the Széchenyi Scientific Society was it possible to perform thousands of experiments through a systematic change in the shape of the shells and to succeed in establishing the basis of haptics, a new branch of prosthetics.

With the rules of haptics, which cannot be listed in detail here, at hand, one does not now select at random among the ready-made shells or bowls and trust the patient's judgment as to the best ones to use; but with the same precision with which the dioptric requirements are prescribed one forms the shells for the individual patient.

Therefore, in the modern prescription for contact glasses one starts with the eye. Each glass is made from a mold and according to the rules of haptics can be checked on the eye itself as to its size, shape, depth and curvatures, and if necessary its form can be corrected. After a perfect shell has been obtained the optimum dioptric effect is given by grinding the outer surface of the center which falls in front of the pupil.

A glass of this type can readily be worn throughout the day, at first with one or two hours of rest at noon. The lenses are always removed at night. The removal or insertion is done by hand and does not require any special skill, but it should always be done with care over a cloth-covered table, because if a lens is dropped from a height on to a hard surface it will break. Lenses that are in contact with the eyeball are better protected than ordinary spectacles, which are easily broken, the splinters often causing serious injuries to the eyes. Most injuries would not directly reach the eyeball itself, and those that do so, e. g., shots, blows with cues and cow horns, destroy the eyeball whether it is covered with a glass shell or not. The shell never cracks spontaneously, and if during the course of years it wears out it can easily be repaired. Its care is simple. In the evening after it is removed it is washed in water, dried with a clean cloth and put away in a padded box.

SUMMARY

The contact eyeglass in its present form is an invisible and permanently wearable thin glass shell, measured and fitted for an individual eyeball according to prescription; it corrects all errors of refraction, including those which spectacles cannot improve. Indications for its use are: (1) irregular astigmatism (keratoconus, corneal scars, maculae, partial leukoma, degeneration or ectasia of the cornea); (2) ametropia and anisometropia of higher degree (high degree of myopia or aphakia); (3) replacement of spectacles because of the wearer's occupation (actor, dancer, orator, pilot, priest, industrial worker) or sport (water sports,

matches, plays) or for cosmetic reasons; (4) protective glass, for instance in cases of incurable entropion, trichiasis and lagophthalmos, or as a light filter for use on the beach, or when crossing snow fields and (5) irreparable weakness of sight (for which an enlarging system can be constructed by combining a simple convex eyeglass with a strong concave shell acting as a mobile ocular of a telescope).

In conclusion I wish to urge that since the contact eyeglass is a prosthesis in touch with a highly sensitive organ, it should be fitted and adjusted with greatest care. The prescribing must be done with due deliberation, but if the use of contact glasses is refused no hesitancy should be shown in recommending them. If a patient is in real need of contact glasses he can accustom himself to them, and his ability to work and to enjoy life will be restored.

ARTIFICIAL FEVER THERAPY IN CASES OF OCULAR SYPHILIS

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In various parts of the world syphilis is held to be responsible for from 4 to 15 per cent of cases of blindness.[1] The various exhibitions of syphilis in the eye rank with those of involvement of the central nervous system in their reluctance to yield to antisyphilitic chemotherapy. The inadequacy of orthodox treatment has been repeatedly demonstrated. Crisp [2] has pointed out that recurrences are common even in patients who have received chemotherapy for many years and who may have had persistently negative serologic reactions.

In 1918 Wagner-Jauregg [3] introduced malaria as a treatment for dementia paralytica, and remarkable results have been obtained by its use in a disease previously regarded as practically incurable. O'Leary and Welsh [4] reported excellent clinical results in approximately 40 per cent of 1,000 cases during a period of nine years. Despite the brilliant results which have been obtained with therapeutic fever following inoculation with infectious agents, such as those of malaria, rat-bite fever and relapsing fever, these methods have serious inherent faults. The

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This investigation was made possible by a grant from the Charles F. Kettering Foundation.

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Presented before the Section on Ophthalmology at the Joint Session of the American Medical Association and the Canadian Medical Association, Atlantic City, N. J., June 14, 1935.

1. Puscariu, E.: Recherches statistiques sur la syphilis oculaire. Fréquence. Séro-diagnostic et signes cliniques extra-oculaires de syphilis. Syphilis ignorée. Syphilis et cécité, Arch. d'opht. **48**:756 (Nov.) 1931. Heller: Statistik der infolge von Geschlechtskrankheiten in Deutschland Erblindeten, Med. Welt. **5**:1583 (Oct. 31) 1931. Clark, T.: Some Public Health Aspects of Syphilis, Ven. Dis. Inform. **12**:205 (May 20) 1931.

2. Crisp, W. H.: The Incurability of Syphilis, Am. J. Ophth. **16**:821 (Sept.) 1933.

3. Wagner-Jauregg, J.: Ueber Einwirkung der Malaria auf die progressive Paralyse, Psychiat.-neurol. Wchnschr. **20**:132 (Aug.) 1918-1919.

4. O'Leary, P., and Welsh, A. L.: Treatment of Neurosyphilis with Malaria: Observations on Nine Hundred and Eighty-Four Cases in the Last Nine Years, J. A. M. A. **101**:498 (Aug. 12) 1933.

ingrafted infection is capable of doing great damage; the mortality in different series ranges from 1 to 10 per cent. It is inconstant in its fever-producing properties. Ten per cent of injections of the malarial virus are unsuccessful, partly because of immunity. There is difficulty in obtaining and maintaining properly attenuated strains of the virus. It is impossible to use antisyphilitic chemotherapy during treatment with malaria. After one malaria treatment it is often impossible again to produce fever with malaria. Malaria treatment requires from two to four weeks of hospitalization.

Typhoid-paratyphoid vaccine and other foreign proteins and sulfur compounds have also been employed for the production of fever. Even though favorable clinical results have frequently followed their use, the febrile response is often inadequate, and severe reactions have occurred.

Neymann and Osborne [5] found that it was possible to induce sustained fever with diathermy. Neymann and Koenig [6] reported that diathermy-induced fever produced a higher rate of remission in patients with dementia paralytica than was obtained in comparable series of patients with the same disease treated with malaria or rat-bite fever. King and Cocke [7] also found that the results of artificially induced fever in unselected patients with dementia paralytica compared favorably with those obtained in selected patients with the same disease treated with malaria. Many other observers have found diathermy to be an effective method for the production of general fever. A wide variety of other physical means have been employed, such as hot baths, hot air, electric blankets and radiothermy.

Fever is primarily an exhibition of a defensive reaction of the body. Reimann [8] stated that fever exerts an adverse influence on the growth of bacteria, diminishes the potency of toxins, favors phagocytosis and stimulates the development of immune bodies. It is believed by some ophthalmologists that this defensive mechanism may be stimulated without pyrexia. This cannot be determined with certainty at present because accurate means for measuring immune reactions are

5. Neymann, C. A., and Osborne, S. L.: Artificial Fever Produced by High Frequency Currents: Preliminary Report, Illinois M. J. **56**:199 (Sept.) 1929; Treatment for Dementia Paralytica with Hyperpyrexia Produced by Diathermy, J. A. M. A. **96**:7 (Jan. 3) 1931.

6. Neymann, C. A., and Koenig, M. H.: Treatment of Dementia Paralytica: Comparative Therapeutic Results with Malaria, Rat-Bite Fever and Diathermy, J. A. M. A. **96**:1858 (May 30) 1931.

7. King, J. C., and Cocke, E. W.: Therapeutic Fever Produced by Diathermy, with Special Reference to Its Application in the Treatment of Paresis, South. M. J. **23**:222 (March) 1930.

8. Reimann, H. A.: Significance of Fever and Blood Protein Changes in Regard to Defense Against Infection, Ann. Int. Med. **6**:362 (Sept.) 1932.

not possessed. There is, however, evidence to indicate that when fever is controlled within its beneficial limits it provides a valuable therapeutic adjunct to chemotherapy in cases of syphilis.

METHOD

Our early experiences with the use of diathermy, radiotherapy, hot baths, electric blankets and infra-red cabinets taught us that these methods possess certain hazards, which are overcome by the use of a simplified air-conditioned cabinet [9] (the Kettering hypertherm) in which the temperature of the air, relative humidity and air velocity are adequately controlled. High frequency electric-currents (diathermy or radiothermy) are no longer employed. With this method a rectal temperature of 105 F. (40.5 C.) or over is usually reached in from forty to sixty minutes. By adjusting the air temperature and humidity, the body temperature may be maintained at the desired level. At the completion of the treatment defervescence is produced by streams of cooler air. The body temperature usually returns to normal in from thirty to forty minutes. The relative simplicity and safety of this method permit us to treat practically all patients without hospitalization.

Most of our patients have been given ten weekly treatments of five hours each at a rectal temperature of from 105 to 106 F. (40.5 to 41 C.). The convincing evidence provided by many observers that fever therapy is most effective when combined with or followed by chemotherapy led us to adopt the practice of giving thirty injections of 0.2 Gm. of bismarsen (bismuth arsphenamine sulfonate). The antisyphilitic drug was given immediately before each of the ten fever treatments, followed by twenty injections at weekly intervals. Each patient had a careful diagnostic survey to determine the eligibility for fever therapy.

Advanced age, myocardial insufficiency and aortic aneurysm were regarded as contraindications. Constant observation by thoroughly trained physicians and nurse-technicians was regarded as an essential feature of the fever treatment. None of our patients suffered any serious effects from the treatments.

Serologic studies were made at weekly or monthly intervals for at least six months, the reactions being studied by two methods (the Kahn and Kolmer tests) in our laboratories and by the Kahn standard diagnostic, presumptive and quantitative methods in the laboratories of R. L. Kahn at the University Hospital at Ann Arbor, Mich. Thereafter, clinical and serologic responses have been recorded at least every six months.

The question has frequently been raised as to how the temperature varies in the deep tissues during fever. In a series of experiments, thermocouples were introduced into the median antibrachial vein, Hunter's canal, the rectosigmoid junction, the cervix uteri, the urinary bladder, the stomach and the orbit. The temperature of the blood rose slightly faster than that of the other tissues during

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9. This apparatus was developed at the Miami Valley Hospital and the Research Laboratories of the Frigidaire Division of the General Motors Corporation, Dayton, Ohio, with the collaboration of Dr. Charles F. Kettering and Mr. Edwin C. Sittler. The apparatus has been described in greater detail by one of us (W. M. S.) elsewhere (J. A. M. A. **105**:2132 [Dec. 28] 1935). Fifty-five of the machines have been lent to twenty medical research institutions for investigative purposes. A simplified form of the apparatus will probably be available ultimately to certain qualified institutions.

the induction of fever, but all the temperatures reached practically the same level as that of the rectum at the end of the first hour and remained at approximately the same level during the five or more hours of maintenance. Ingestion of ice-water caused fluctuation of the temperature of the stomach but did not affect that of the other tissues. Continuous ice-cold compresses to the eye for a period of seventy minutes during the height of fever produced a reduction in the temperature of the orbit at a depth of 38 mm. of less than 0.5 F. and did not affect the temperature of the other deep tissues (fig. 1). These values for deep temperature are essentially similar to those found during fever induced by high frequency methods.

CLINICAL DATA

During the past four years we have subjected 383 patients to artificial fever therapy. Of these, 175 patients were treated for syphilis. Our aim has been to give each patient with syphilis at least fifty hours

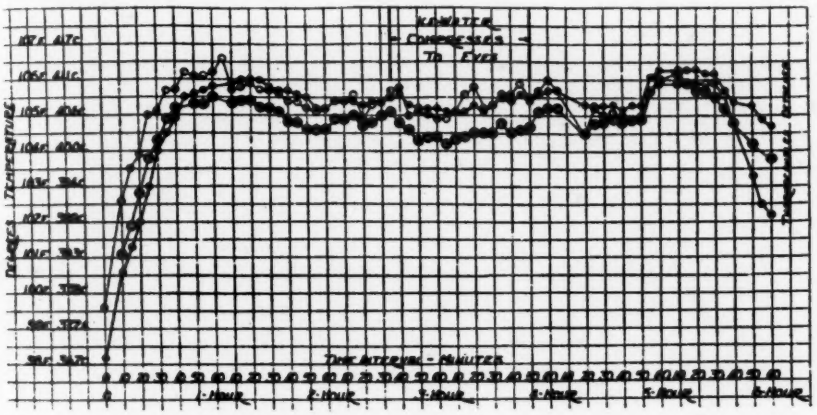

Fig. 1.—Chart showing deep tissue temperatures of the orbit, circulating blood of the median antibrachial vein and rectum (depth of 12 inches [30.5 cm.]), based on thermocouple determinations made at five minute intervals for a period of six hours. In this chart the temperature of the rectum is indicated by a line of dots; that of the blood in the median antibrachial vein, by a line of small hollow circles, and that of the orbit, by a line of double circles.

of sustained fever therapy usually divided into ten weekly treatments consisting of five hours or more of fever during which the rectal temperature was from 105 to 106 F. (40.5 to 41.1 C).

Fifty-eight of the patients with syphilis had an ocular complication of the disease. We have excluded the cases in which only changes in the pupillary reflexes or the scars of old inactive lesions were shown. In 4 of the 58 cases of ocular syphilis the condition had a double classification, as follows: conjugate palsy and atrophy of the optic nerve, uveitis of the right eye and atrophy of the left optic nerve, uveitis of the right eye and neuroretinitis of the left and neuroretinitis of the right eye and choroiditis of the left.

In the 58 cases diagnosis of the primary conditions was made in 62 eyes, as follows: extra-ocular palsy in 4; interstitial keratitis in 11; exudative uveitis in 10; optic neuritis and neuroretinitis in 14; choroiditis in 7, and atrophy of the optic nerve in 16.

In the 116 eyes additional changes due to syphilis were shown. The associated secondary conditions were: residual traces of old uveitis, such as synechiae, uveal cataract and opacities of the vitreous in 4 instances, old choroiditis in 6, interstitial keratitis in 7, gumma of the iris in 1, and pupillary changes, including the Argyll Robertson pupil, in 24.

The clinical data in each group are tabulated, and the course of the disease in representative cases of each group is summarized.

Group 1: Extra-Ocular Palsy (4 Cases).—Four patients were treated: 2 with palsy of the sixth nerve, 1 with ptosis and palsy of the superior rectus muscle and 1 with conjugate palsy. In 2 cases of comparatively recent syphilis improvement was shown and in 2 of long standing no improvement occurred.

TABLE 1.—*Clinical Data in 4 Cases of Extra-Ocular Palsy* *

Case	Type	Duration	Previous Treatment	Clinical Results
1	Sixth nerve	3 months	None	Orthophoria
2	Sixth nerve	2 years	2 years	Fusion to 20° temporally
3	Third nerve	10 years	23 years	No improvement
4	Conjugate	14 years	14 years	No improvement

* The average period of observation was twenty months.

E. L., a salesman aged 44, complained of diplopia on looking to the left, of two years' standing. The serologic reactions of the blood and spinal fluid were positive, and the patient had received neoarsphenamine, bismuth and iodides in alternation for two years without clinical improvement. On examination, with the right eye in the primary position, the left eye deviated mesially 26 degrees, and the patient was unable to rotate it to the left beyond the primary position. Homonymous diplopia was present in all fields but increased markedly to the left. Argyll Robertson pupils were present, the left being slightly larger than the right. Examination of the fundus showed only well marked periarteritis. The diagnosis was complete paralysis of the left sixth nerve, with secondary deviation. The patient was given fifty hours of fever therapy with a temperature of from 105 to 106 F. (40.5 to 41.1 C.) at weekly intervals and thirty weekly injections of bismarsen. After the tenth treatment, no change was noted in the pupils or the field of diplopia, the diplopia on the left still being too great to chart. The active periarteritis, however, had given place to variable white lines along the vessels. Eight months after treatment was started, the pupils were equal and reacted sluggishly to light, and the patient was able to rotate the left eye to a position 30 degrees temporally. He was, however, suppressing this eye, and he was given fusion exercises with the stereoscope. After one month of these exercises the secondary deviation disappeared, and he had stereoscopic vision to 20 degrees to the left. The Kahn and Kolmer reactions of the blood serum and spinal fluid were reversed to negative.

Group 2: Interstitial Keratitis (11 Cases).—Eleven patients with interstitial keratitis, ranging in age from 8 to 38 years, received fever therapy and bismarsen. Eight of the 11 patients had had previous attacks. Seven of these attacks occurred while the patient was receiving active antisyphilitic therapy. One severe condition showed no response to six weekly injections of neoarsphenamine, and the patient recovered with fever therapy. All but 1 patient had had previous therapy of various intensity. The patients, at the time of writing, have been under our observation for an average period of eighteen months. Of the 20 eyes involved in 11 patients, none have less than 6/30 vision, and 18 have 6/12 vision or better.

There has been only 1 instance of recurrence in this group. We observed this exceptional patient during four attacks for a period of two years before the fever treatments. He has had only one attack

TABLE 2.—*Clinical Data in 11 Cases of Interstitial Keratitis*

Previous attacks	None 3	One 4	Two or More 4
Previous antisyphilitic treatment	None 1	Moderate 1	Intensive 9
Final vision (20 eyes)	Vision less than 6/60 0	Vision from 6/60 to 6/15 2	Vision from 6/12 to 6/5 18
Recurrences	None 10	One 1	Two or More 0
Duration of attacks	1 to 4 months		
Average period of observation	18 months		

in thirty months since the treatments, although the course was incomplete. The average duration of an attack from the beginning of observation until the disappearance of ciliary injection was about two months.

R. Z., a boy 8 years of age, was admitted to the hospital because of severe photophobia and loss of vision of three weeks' duration. He had Hutchinson teeth and the physiognomy characteristic of congenital syphilis. The Kahn and Kolmer reactions of the blood were strongly positive, while the findings in the spinal fluid were normal. In spite of active local and systemic treatment, the patient lay with his head buried under a pillow in a darkened room for six weeks. We were able to make the first careful examination of the eyes on the day after the first fever treatment. At that time there was marked ciliary injection, and confluent dirty grayish plaques mottled the cornea of each eye. Roughly crescentic masses of plastic precipitates occupied the pericentral region of both eyes. The iris could be seen faintly. The exudate showed perceptible diminution after each treatment. The maculae in the corneas changed very slowly. After the eighth weekly fever treatment the photophobia and evidence of active inflammation had disappeared. The vision had improved to 3/60 and 6/60. Six months later the vision had improved to 6/60 and 6/30, and to 6/15 in both eyes with refraction. After twenty-nine months, the eyes showed interstitial vascularization and numerous

maculae of the corneas and scattered tiny atrophic patches in the choroid. There was no residual trace of the plastic precipitates; the pupils were active, and there were no other opacities in the media. Visual acuity was 6/12 —1 and 6/12 —2 with lenses. The patient had had no recurrence in twenty-nine months. There was also remarkable improvement in the mental state.

Group 3: Exudative Uveitis (10 Cases).—Ten patients, aged from 22 to 45 years, were treated. In 6 instances exudative uveitis occurred during active antisyphilitic therapy. In 2 instances it occurred after considerable antisyphilitic treatment. One patient showed exacerbation with neoarsphenamine therapy. Two failed to improve after intravenous injections of typhoid vaccine. In 7 of the patients there was so much exudate in the anterior segment that it was impossible to make out details of the fundus. This group is composed of patients who had failed to respond to the usual antisyphilitic therapy, and serious

TABLE 3.—*Clinical Data in 10 Cases of Exudative Uveitis*

Previous treatment	Antisyphilitic 8 (5 wks. to 10 yrs.)		Typhoid Vaccine 2 (3 to 6 wks.)
Vision at onset (12 eyes involved)	Vision less than 6/60 5	Vision from 6/60 to 6/15 7	Vision from 6/12 to 6/5 0
Appearance of clinical improvement	After one treatment 8	After two treatments 2	After three or more treatments 0
Condition quiescent	After one to three treatments 5	After four to six treatments 3	After seven to ten treatments 2
Final vision (11 eyes, results in 1 unrecorded)	Vision less than 6/60 1	Vision from 6/60 to 6/15 0	Vision from 6/12 to 6/5 10
Recurrences	None 9	One 1	Two or more 0
Average observation	17 months		

impairment of vision seemed inevitable. All showed prompt and striking clinical improvement during fever therapy. After one or two treatments there was distinct diminution in the degree of ciliary injection, decrease in the amount of exudate, improvement in the dilatation of the pupil and relief of symptoms. The progress in most cases was rapid, leaving few residual defects. In 1 case a slight increase in the amount of exudate in the eye was twice shown on the day after a fever treatment. However, the condition of the eye was improved each time before the end of the following week. This is also the only case in which the patient had a mild recurrence of the uveitis during an average interval of observation of seventeen months. This patient was receiving additional fever treatments at the time of writing. Except for 1 patient, who experienced exacerbation of the condition in an eye which already showed extensive degenerative changes, all the subjects recovered useful vision.

W. B., a plumber aged 26, had a chancre of the penis three months before his first visit. After he had received neoarsphenamine therapy for seven weeks, blurring of vision developed, with redness of the right eye but no pain. Vision was 1/60 in the right eye and 6/7.5 in the left. Examination revealed exudative uveitis in the right eye and low grade neuroretinitis in the left. The patient was given atropine locally. Bismuth and iodides were administered by the physician. The right eye improved slowly. Later the patient was given two courses of neoarsphenamine with injections of bismuth between the courses. Five months after the first attack he returned with marked pain, photophobia, ciliary injection, diffuse grayish infiltration of the lower part of the cornea with three linear folds in Descemet's membrane, keratic precipitates, gross engorgement of the iris and exudate in the pupillary space of the left eye. A pinkish nodule, 1.5 mm. in diameter, arose from the base of the iris of the left eye in the position of 9 o'clock. Examination of the fundus was impossible. Vision was 6/7.5 in the right eye, and the patient was able to count fingers at 3 feet (91.44 cm.) with the left. The day after the first fever treatment the eye showed much less exudate. It was possible to see some details of the fundus. The nodule had not changed. After a course of fifty hours of fever treatment with a temperature above 105 F. (40.5 C.) combined with chemotherapy, vision was 6/7.5 in the right eye and 6/7.5 in the left. The nodule had disappeared. The pupillary reflex was active. A few fine flakes of pigment adherent to the anterior capsule were the only gross residual defect. The patient has had no recurrence in ten months. The serologic reaction of the blood and spinal fluid were reversed to negative.

J. E., a Negro chauffeur aged 38, had consulted an ophthalmologist on April 5, 1933, because of severe exudative uveitis of the right eye. The Kahn and Kolmer reactions of the blood serum and spinal fluid were strongly positive. The patient was treated with atropine and given three courses of neoarsphenamine. The uveitis responded to this treatment. The serologic reactions were still positive, and the patient was receiving active antisyphilitic chemotherapy when he returned on Dec. 8, 1933, with marked epiphora, ciliary injection, several discrete grayish plaques deep in the stroma of the cornea, marked engorgement of the irides with grayish exudate on the pupillary border and extensive posterior synechiae in both eyes. Vision was 6/30 in each eye. Details of the fundus were too blurred for description. With intensive local treatment most of the synechiae were broken. The patient was given fifty hours of therapeutic fever combined with chemotherapy. There were less injection and exudate, and relief from pain and photophobia was obtained on the day following the first five hour fever treatment. After the sixth weekly fever treatment the corneas were clear. All evidence of active inflammation had disappeared. The pupils were partially dilated. A bandlike synechia was present mesially in the right eye, and two smaller synechiae were observed in the left. Scattered pigment dots were present on the anterior capsule. Vision with the use of atropine was 6/12 in the right eye and 6/15 in the left. The fundus was normal except for faint white lines along some of the vessels. Vision at the time of writing was 6/12 in the right eye and 6/7.5 in the left, and there had been no recurrence in sixteen months. The Kahn and Kolmer reactions of the blood serum became less strongly positive, and the reactions of the spinal fluid became negative.

C. S., a white man aged 24, acquired a chancre of the penis in August 1932. He had received two courses of neoarsphenamine and weekly injections of bismuth. When he was seen on Dec. 15, 1932, there were marked photophobia and ciliary injection of the right eye. There were a few faint grayish folds in Descemet's membrane, scattered keratic precipitates and gross engorgement of the iris; the

individual vessels were visible in the stroma of the iris, and posterior synechiae remained in the positions of 3 and 9:30 o'clock after intensive atropine therapy. There were coarse grayish opacities in the vitreous, and details of the fundus were not made out. Vision was 5/60 in the right eye and 6/15 in the left. The left eye showed a macula of the cornea lying temporally from the visual axis and neuroretinitis. The patient was given a course of fever treatment combined with chemotherapy. Improvement was noted after the second fever treatment. The right pupil, which was only partially dilated by intensive administration of atropine and repeated subconjunctival injections of epinephrine, became widely dilated. On Feb. 14, 1933, after completion of fifty hours of fever therapy and chemotherapy, vision was 6/7.6 —2 in the right eye and 6/12 —1 in the left. All evidence of active inflammation in the right eye had subsided, leaving synechiae in the positions of 3 and 9:30 o'clock, stringy opacities in the anterior part of the vitreous and slight blurring of the rings. The left fundus was normal. On March 9, 1934, refraction of the eyes was again determined, and vision was improved to 6/6 in both eyes. Vision had remained normal since that time. The Kahn and Kolmer reactions of the blood serum and spinal fluid became negative in seven months and remained so.

TABLE 4.—*Clinical Data in 14 Cases of Optic Neuritis and Neuroretinitis*

Previous treatment	Intensive 8	Moderate 4	None 2
Vision at onset of therapy (26 eyes involved)	Acuity less than 6/60 1	Acuity from 6/60 to 6/15 10	Acuity from 6/12 to 6/5 15
Final vision	0	0	26
Residual contraction of visual fields	More than 15° 1	15° to 5° 15	None 10
Relapses	None		
Average period of observation	19 months		

Group 4: Optic Neuritis and Neuroretinitis (14 Cases).—This group includes 2 patients who presented evidence of secondary manifestations of syphilis. In 1 instance this occurred during active treatment with arsenicals and bismuth. In the other patient, the cutaneous lesions appeared after the first injection of neoarsphenamine. In 8 cases the ocular lesions were associated with involvement of the central nervous system. All but 2 patients had had considerable previous chemical treatment. All the patients at the time of writing have useful vision (6/12 or more), although 8 have residual pallor of the disk and slight contraction of the visual fields.

T. C., a housewife aged 30, complained on Jan. 27, 1933, of increasing dimness of vision for six days. Vision was 6/15 in the right eye and 1/60 in the left. The right fundus showed slight neuroretinitis, and the left, gross neuroretinitis. The disk was swollen 2 diopters. The optic cup was nearly obliterated. The rings were entirely obscured. Whitish edema of the retina extended throughout the central portion of the fundus, involving the macula and partially obscuring the vessels. The patient was given typhoid vaccine intravenously. The Kahn

and Kolmer reactions of the blood were strongly positive. However, the eye looked considerably better three days later, and the scotoma had decreased in size. The patient was given another injection of typhoid vaccine on that day. On Feb. 6, 1933, she received an injection of neoarsphenamine, after which the edema and scotoma increased. A course of fever treatments combined with bismarsen was administered. With the first fever treatment vision improved during the next twenty-four hours from 3/60 to 6/30. At the completion of the treatment, vision was 6/30, and with correction, 6/12. The changes in the scotoma, based on studies with the tangent screen, are shown in figure 2. The fundus showed residual connective tissue over the nerve head, white lines along the

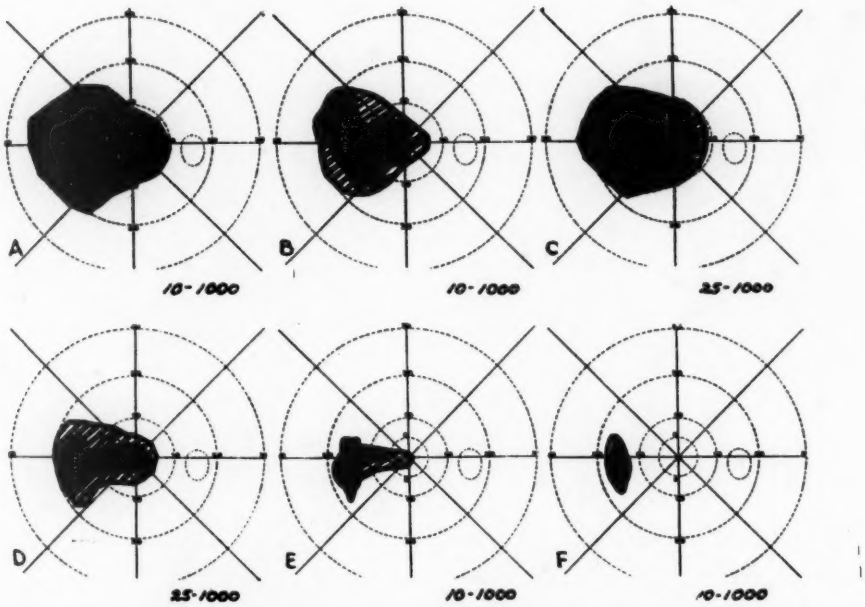

Fig. 2.—Changes in the central scotoma in a case of syphilitic neuroretinitis of the left eye, showing (*A*) a large absolute scotoma involving the entire central zone found at the first observation (Jan. 27, 1933), (*B*) diminution in the size of the scotoma following two intravenous injections of typhoid vaccine, with the relative scotoma indicated by the shaded area (February 3), (*C*) enlargement of the scotoma four days after an injection of neoarsphenamine (February 10), (*D*) marked diminution in the size of the scotoma twenty-four hours after the first fever treatment, associated with decrease in the edema of the fundus (February 11), (*E*) further diminution in the size of the scotoma after the third fever treatment, then almost entirely relative (February 25) and (*F*) slight blurring of the rings and the corresponding relative scotoma after the seventh fever treatment (March 25). The central fields were normal at the completion of the last (tenth) fever treatment.

arteries and a slight granular change in the macula. It is possible that this patient might have done as well with typhoid vaccine therapy, but she was exhausted after two treatments with the vaccine. She tolerated the fever treatments well.

Group 5: Choroiditis (7 Cases).—In all of the 7 cases choroiditis had appeared, reappeared or progressed with the usual antisyphilitic chemotherapy. The lesions responded promptly to fever therapy. The exudate disappeared in an average period of six weeks, leaving residual defects in the fields. Fortunately, none of the lesions involved the macula, and good central vision was obtained in all the eyes but 1, which had 6/30 vision. It is noteworthy that the lesions progressed in 2 patients whose serologic reactions had been reversed to negative with previous chemotherapy. In an average period of observation of twenty-four months we noted no exacerbation of choroiditis following fever therapy combined with chemotherapy.

TABLE 5.—*Clinical Data in 7 Cases of Choroiditis*

Previous treatment.....	Intensive 7	Moderate 0	None 0
Vision before fever treatment (13 eyes involved)	Vision less than 6/60 2	Vision from 6/60 to 6/15 10	Vision from 6/12 to 6/5 1
Final vision.....	0	1	12
Residual defects of the field.....	13		
Average interval of observation.....	24 months		
Recurrences.....	None		

TABLE 6.—*Clinical Data in 16 Cases of Atrophy of the Optic Nerve*

Previous treatment.....	Intensive 9	Moderate 6	None 1
Contraction of visual fields (32 eyes)	More than 30° 16	30° to 15° 9	15° or less 7
Change in fields.....	Improvement 6	Loss 6	No change 20
Average interval of observation.....	20 months		

B. D., aged 23, complained of "seeing cobwebs before the left eye." He had received continuous antisyphilitic therapy during the preceding two years. During the past year he had experienced two exacerbations of blurring of vision, each attack leaving residual diminution in acuity. Examination of the fundus revealed old diffuse choroiditis in the right eye, and in the left, fine clouds of dustlike opacities in the vitreous and a few coarse strings. There was a broad diffuse patch of choroiditis in the superior part of the temporal portion of the periphery, while the periphery below showed a broad yellowish edematous area with a fuzzy border, in which all details were blurred. Vision was 6/30. The patient was given a course of fever treatments combined with chemotherapy. After the second treatment the vitreous was about the same as before therapy, but the borders of the area of choroiditis were more sharply defined. After the fourth treatment the choroiditis became quiescent, leaving a residual defect in the field. Vision was 6/12—1. The field remained practically constant after treatment. There was no recurrence in thirty-eight months. The Kahn and Kolmer reactions of the blood serum and spinal fluid remained negative.

Group 6: Atrophy of the Optic Nerve (16 Cases).—The patients in this group, with 1 exception, had received previous antisyphilitic therapy. In several cases vision had been temporarily improved by previous treatment. In 1 case vision had failed during malaria

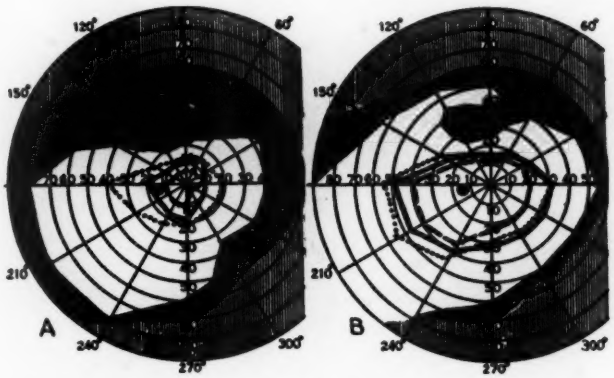

Fig. 3.—Perimetric studies showing (*A*) defects in the field due to active disseminated choroiditis of the left eye (Jan. 29, 1932) and (*B*) the residual defects remaining on March 4, after four fever treatments. This field has persisted practically unchanged for thirty-seven months after the completion of combined fever therapy and chemotherapy. The visual fields shown in *A* and *B* were obtained by a 5 mm. test object at 300 mm. Vision when the field shown in *A* was taken was 6/30 and when the field shown in *B* was taken, 6/12. In this figure the field for blue is outlined by a solid line; the field for red, by a line of dots, and the field for green, by a line of dashes.

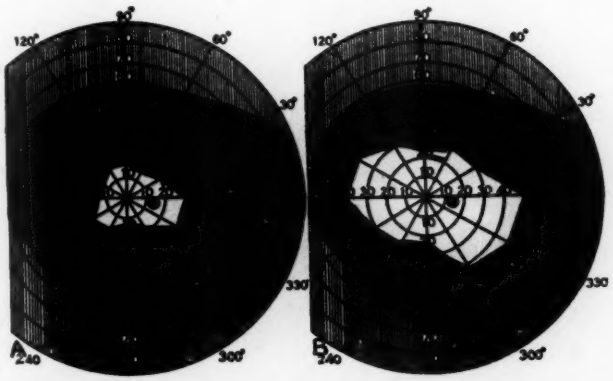

Fig. 4.—(*A*) form field in a case in which atrophy of the right optic nerve was shown, taken on March 4, 1935, before fever treatment, and (*B*) the field for the same eye, taken eight months after fever therapy was instituted (November 9). Field *B* remained unchanged. The visual fields shown in *A* and *B* were obtained by a 12 mm. test object at 300 mm. Vision when the field shown in *A* was taken was 3/60 and when the field shown in *B* was taken, 3/60.

therapy. In another case vision had failed after tryparsamide therapy. After fever therapy the central vision remained practically unchanged. The visual fields showed slight improvement in 3 cases, slight diminution in 3 and practically no change in the other 10 during an average period of observation of twenty months.

F. R., a laborer aged 40, complained of increasing dimness of vision for two years. He had received antisyphilitic chemotherapy during this interval, with slight improvement in vision at the beginning of the course of treatment. Vision had been practically stationary for a year. Acuity was 3/60 in the right eye. Moving shadows could be detected with the left eye. The principal complaint was increasing difficulty in walking. Argyll Robertson pupils were present. The right fundus showed marked pallor of the disk, with a large cup and prominent lamina cribrosa. The rings were distinct, except for slight blurring above and below. The arteries were contracted. In the left fundus the whole disk showed saucer-like cupping and was glistening white. The arteries were markedly contracted. The visual field showed slight improvement with fever therapy, and this has been maintained for a year. We saw no evidence of active neuritis in the fundus, but there must have been an active inflammatory process at some point along the optic tract.

COMMENT

Extra-Ocular Palsy.—The results obtained with fever therapy are comparable to those obtained with other methods of treatment. Patients with recent involvement sometimes recover completely with iodides alone. Paralysis established for years is probably stationary. The evidence adduced from these studies shows that some return of function is possible even after palsy has existed for two years.

Interstitial Keratitis.—Evaluation of therapeutics in interstitial keratitis is particularly difficult because of the great variability in the severity and length of the course of acute attacks and in the density of subsequent opacities. Hutchinson first established the association of interstitial keratitis and congenital syphilis in 1855. Spicer,[10] in a comprehensive monograph, reported his observations in 659 cases from the same clinic at Moorfields. In 45 of Spicer's cases no antisyphilitic treatment was received. He stated: "They did not compare unfavorably with others in which mercury had been continuously used. . . . In the great mass of cases, the arsenical compounds have no effect, though now and then remarkable improvement follows their use." He did not use arsenicals except in cases of severe involvement.

Fisher [11] expressed skepticism as to the possibility of modifying the course of the disease by any antisyphilitic treatment, though most

10. Spicer, W. T. H.: Parenchymatous Keratitis: Interstitial Keratitis: Uveitis Anterior, London, P. Pulman & Sons, 1924.

11. Fisher, J. H.: Discussion of the Value of Recent Methods of Treatment in the Late Stages of Ocular Syphilis, Proc. Roy. Soc. Med. **20**:954, 1927.

observers attribute a favorable influence to the use of arsenicals, mercury and bismuth. Foreign protein therapy has been used successfully by Marchesani,[12] Huber,[13] Cowley[14] and others.

The malaria treatment of interstitial keratitis was first described by Schreiber,[15] who reported 23 cases and made the observation that the course of the disease was shortened and that the pupil dilated more readily, though scarring of the cornea was practically as severe as with other methods of treatment. Albrich,[16] Granström[17] and Meller[18] reported favorably on the results of malaria therapy. Dennie[19] reported 8 cases of interstitial keratitis which "responded in from one-fourth to one-half the time that it ordinarily takes to clear up this type of case." Ambler and Van Cleve[20] treated 17 patients with malaria and reported "rapid and complete cessation of pain, photophobia and lacrimation." While, unfortunately, the results in respect to vision were not recorded, none had residual impairment of vision sufficient to cause an industrial handicap. Two recurrences were noted after the treatment. Clapp[21] expressed the belief that inoculations with the malarial virus were too dangerous to justify further trial, although he recommended the use of local treatment with roentgen radiation in the early stages.

This great difference of opinion is due partly to the lack of statistics dealing with the results on vision. These are indeed difficult to determine, as years of quiescence bring increased transparency to a vascularized cornea. James[22] reported recovery of vision of 6/12 or better

12. Marchesani, O.: Beitrag zur unspezifischen oder Fiebertherapie der Keratitis parenchymatosa, Arch. f. Augenh. **99**:207 (May) 1928.

13. Huber, R.: Keratitis parenchymatosa, Klin. Monatsbl. f. Augenh. **75**:252, 1925.

14. Cowley, A. H.: Some Observations on the Treatment of Interstitial Keratitis, Kentucky M. J. **30**:423 (Aug.) 1932.

15. Schreiber, L.: Treatment of Keratitis Parenchymatosa with Malaria, Ztschr. f. Augenh. **66**:316, 1928.

16. Albrich, K.: Ueber den Einfluss von Infektionskrankheiten auf Augenleiden, Klin. Monatsbl. f. Augenh. **81**:61 (July 27) 1928.

17. Granström, K. O.: Malarial Treatment of Parenchymatous Keratitis, Hygiea **92**:830 (Nov. 15) 1930; cited by Ambler and Van Cleve.[20]

18. Meller, J.: Welchen Wert hat die Malariatherapie bei luetischen Augenerkrankungen? Aerztl. Praxis **5**:173 (June 15) 1931.

19. Dennie, C. C.: Effect of Malarial Therapy upon the Congenital Syphilitic, J. Missouri M. A. **29**:581 (Dec.) 1932.

20. Ambler, J. V., and Van Cleve, J. V.: Malarial Therapy in Syphilitic Interstitial Keratitis, J. A. M. A. **102**:1553 (May 12) 1934.

21. Clapp, C. A.: Interstitial Keratitis, Arch. Ophth. **2**:580 (Nov.) 1929.

22. James, W. M.: Stovarsol in the Treatment of Ocular Syphilis, J. Missouri M. A. **30**:33 (Jan.) 1933.

in 7 of 9 eyes involved in 6 cases in which treatment with acetarsone was carried out. The exhaustive study of interstitial keratitis by Carvill and Derby [23] showed a resultant vision of 6/20 or better in 75 per cent of 100 treated patients and in 56 per cent of 100 patients not treated. With the collaboration of Dr. A. H. Benz, of the University of Michigan, we tabulated for comparison the results in 25 cases of interstitial keratitis in which treatment with arsphenamine and mercury was given. These cases were selected on the basis of the known final vision, because the eyes had been refracted. Of the 50 eyes, the vision was less than 6/60 in 4 (8 per cent), from 6/60 to 6/15 inclusive in 24 (48 per cent) and from 6/12 to 6/5 inclusive in 22 (44 per cent).

Twenty eyes were involved in the 11 patients in our series. Since 8 of the patients had recurrent keratitis and 10 were selected on the basis of the failure of orthodox therapy, they were not an average group. Results on vision in a larger series may show considerable variation in either direction from our findings. As a matter of record, the visual acuity in no eye was under 6/60, although a patient receiving treatment at present may not recover much vision. Ten per cent of the patients have obtained corrected vision of from 6/15 to 6/60 inclusive, and 90 per cent have visual acuity of 6/12 or more.

Comparison of individual cases in our personal experience leads us to stress the importance of an observation made by Spicer. The attacks which especially endanger vision are neither those in which gross vascularization is shown nor the recurrent type. The most alarming sign is an opaque central disk of plastic precipitates. If this remains long it destroys the endothelium and leaves the center of the cornea permanently and densely opaque. This is the type which shows immediate improvement with sustained fever, as the exudate is usually diminished after each fever treatment. No such remarkable change occurs in the corneal maculae. Perusal of the literature leads us to suspect that this diminution of exudate has also been seen after malaria therapy and with injections of typhoid vaccine and that it accounts for the enthusiasm in some of the reports on these forms of therapy.

On the other hand, in the cases in which little or no invasion of cellular exudate is shown, except in the stroma of the cornea itself, only gradual improvement appears. The question as to whether corneal maculae clear more rapidly and more completely with than without fever therapy cannot be answered without the influence of the personal equation, except by the study of large series of cases with complete records on vision.

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23. Carvill, M., and Derby, G. S.: Interstitial Keratitis, Boston M. & S. J. **193**:403, 1925.

Spicer observed recurrences in 9 per cent of his series of 659 cases, while the statistics of Carvill and Derby [23] showed relapses in 3.6 per cent of 100 cases in which treatment was given and in 27 per cent of cases in which it was not given. Our observations in a group of 8 cases of recurrence, in which only 1 relapse appeared during eighteen months, lead us to believe that the tendency to recurrence is diminished by fever therapy.

The average duration of an attack from the first appearance of photophobia and ciliary congestion to its entire disappearance was four months in Spicer's series and six months in Downing's.[24] In the 11 cases in our series, the disease ran a course of from one to four months.

Exudative Uveitis.—The immediate clinical response of this disease to artificial fever therapy has been uniformly favorable. Mild recurrence occurred in 1 case, probably because of inadequate fever therapy. All but 1 of the 10 patients recovered reading vision. It is our opinion that the course of the disease and the residual defects have been diminished in all these cases and that fever therapy combined with chemotherapy is the treatment of choice for syphilitic exudative uveitis.

Optic Neuritis and Neuroretinitis.—Immediate diminution in the amount of exudate in the fundus is observed after fever therapy in these conditions, as it is in uveitis. There seems to be arrest in the optic neuritis associated with syphilis of the central nervous system after fever therapy. The permanence of this effect has not yet been established.

Choroiditis.—In active choroiditis with exudate there is usually subsidence of the lesion with fever therapy, though this is not as rapid as in uveitis.

Atrophy of the Optic Nerve.—Neymann and Osborne [25] described 6 cases of syphilitic atrophy of the optic nerve in which a minimum of twenty fever treatments were given, the temperature in each ranging from 105.8 F. (41 C.) to 107.6 F. (42 C.) for six hours. In all these cases the condition was advanced, and the findings in the spinal fluid were positive. While the patients were under observation less than two years, vision was entirely lost in 2, unchanged in 2 and improved in 2.

In our experience visual acuity and visual fields in cases of atrophy of the optic nerve have been practically unchanged by fever therapy, an observation common to cases in which treatment with malaria was

24. Downing, J. A.: Interstitial Keratitis: Treatment, Results and Case Reports, J. Iowa M. Soc. **15**:60 (Feb.) 1925.

25. Neymann, C. A., and Osborne, S. L.: Development of Hyperpyrexia, Arch. Phys. Therapy **15**:149 (March) 1934.

given, as reported by Fleischer.[26] Possibly the progression of atrophy is somewhat retarded.

In this connection we see no reason for the assumption that atrophy of the optic nerve is in itself progressive. We have seen vision in cases of atrophy of the optic nerve due to various causes remain stationary for years. Indeed, the common history of patients with atrophy due to tabes shows great variation in the progressive loss of vision, with temporary improvement under some therapeutic measures, long intervals of apparent quiescence and periods of progression. This can mean only that the progression corresponds to periods of active syphilitic infection of the optic tract. Therapy capable of halting other forms of involvement of the central nervous system should have a similar influence on the visual areas of the brain. That this has not been observed with some forms of antisyphilitic therapy may be due in part to the fact that gradual loss of function of some parts of the brain may not be immediately apparent, while the visual apparatus affords opportunity for close and comparative study. Slight changes in vision are manifest even to the patient himself.

Our series of cases is small, and the average period of observation was only twenty months, but in the majority of instances the atrophy did not progress during this interval. The results of serologic studies showed that syphilis in most of the patients was only quiescent. Further therapy will undoubtedly be required. However, if in larger series a comparable quiescence of the syphilis of the optic tract is shown with this treatment, a real advance has been made. One case illustrates the point: Six months ago, this patient's vision had remained unchanged for thirty-three months after fever therapy combined with chemotherapy. On account of positive serologic reactions he had received extensive chemotherapy during this interval. The visual fields at the last taking showed a slight contraction as compared with those at the preceding one. At the time of writing the patient was receiving another course of fever treatments, the outcome of which we anticipate with interest. It is apparent that atrophy of the optic nerve is not a contraindication to fever therapy.

Associated Diagnoses.—Residual changes due to previous attacks of uveitis, such as posterior synechiae, uveal cataract and opacities of the vitreous remained unchanged by the treatments. The same observation was made in cases of the opacities and vascularization of old interstitial keratitis and of the scars of old choroiditis.

In most cases of exudative uveitis coarse precipitates were shown on the posterior surface of the cornea and, in addition, edema of the deeper

26. Fleischer, B.: Fever Treatment of Tabetic Atrophy of the Optic Nerve, Klin. Monatsbl. f. Augenh. **90**:335, 1933.

layers of the cornea. Folds in Descemet's membrane (striate keratitis) were present in 6 cases. Interstitial keratitis secondary to the uveitis occurred in 5 instances. These complications improved with subsidence of the primary process, although the keratitis left faint maculae in 1 case.

Changes in the pupillary reflexes, including Argyll Robertson pupils, were observed in 34 of the 58 patients. All the 34 patients had some form of syphilis of the central nervous system. In 3 cases the condition was associated with extra-ocular palsy, in 5 with choroiditis, in 10 with optic neuritis and in 16 with atrophy of the optic nerve. Unequal pupils became equal in 3 cases. There was some return of the light reflex in 2 instances. In the others practically no change was shown.

SUMMARY AND CONCLUSIONS

Ocular syphilis is a common cause of blindness. The remarkable results which have been achieved with various forms of artificial fever therapy in the treatment of neurosyphilis led us to undertake a study of the influence of fever therapy, combined with antisyphilitic chemotherapy, on ocular syphilis.

The failure of ocular syphilis to respond to chemotherapy in many cases may be compared to the frequent failure of chemotherapy to exert a favorable influence on neurosyphilis.

Favorable results have been obtained in the treatment of neurosyphilis with a wide variety of infectious, protein, chemical and physical agents. The one factor common to all is the production of sustained fever.

With the utilization of a comparatively simple, safe and controllable air-conditioned apparatus for the induction and maintenance of fever (Kettering hypertherm), 58 patients with various manifestations of ocular syphilis have been treated with approximately fifty hours of fever with temperatures above 105 F. (40.5 C.) in ten weekly sessions of five hours each. A course of thirty injections of bismarsen was given in conjunction with the fever treatments.

Most of the patients with ocular syphilis who were chosen for this study had failed to respond to orthodox therapy.

In the evaluation of the response of 4 patients with extra-ocular palsy to artificial fever therapy there was no evidence that this method is superior to older forms of treatment.

Of the 11 patients with interstitial keratitis, 8 had experienced recurrences, and 10 had failed to respond to chemotherapy. The duration of the disease and the tendency to recurrence appear to be distinctly lessened after adequate fever therapy combined with chemotherapy. The response is most prompt in the cases in which an opaque central

disk of plastic exudate exists. It is usually this type of the disease which produces the greatest visual damage.

In 10 cases of exudative uveitis prompt clinical improvement became apparent in every instance after the first one or two fever treatments. With the exception of 1 patient who had advanced old degenerative changes, all recovered useful vision. One patient experienced mild recurrence, probably because of inadequate fever therapy. With due regard to the small number of cases in this series, the results were so uniformly favorable that we regard fever therapy combined with chemotherapy as the treatment of choice in syphilitic exudative uveitis.

Favorable response occurred in the lesions of 14 patients with optic neuritis and neuroretinitis. All the patients recovered useful vision although 9 have residual pallor of the disk and some degree of contraction of the fields. Active neuritis of the optic tract appears to be arrested by fever therapy.

Active choroiditis in 7 patients subsided with fever therapy, with residual scars and defects of the fields. Good central vision resulted in all but 1 eye.

In 16 patients with atrophy of the optic nerve the visual acuity and visual fields remained practically unaltered after a course of fever therapy combined with chemotherapy. If atrophy of the optic nerve is associated with foci of active infiltration along the optic tract, it is probable that such exudative lesions will respond to fever therapy. Atrophy of the optic nerve is not a contraindication to fever therapy.

Artificial fever therapy is most effective in cases of the exudative lesions of ocular syphilis.

As in neurosyphilis, there is no parallelism between the clinical and the serologic response of syphilitic ocular disease to fever therapy combined with chemotherapy.

There is evidence which strongly suggests that the greatest field of usefulness for artificial fever therapy combined with specific therapy will ultimately be in its application to the early manifestations of syphilis, with a view to prevention of the often disastrous ocular complications.

ABSTRACT OF DISCUSSION

Dr. William L. Benedict, Rochester, Minn.: When heat is produced generally, as in fever, there is, in addition to its local action, a physiologic factor that makes available certain nonspecific antibodies, which in themselves probably exert a favorable influence.

The use of hot baths and moist air chambers to raise the body temperature to 105 F. for five or six hours is an innovation and while several of the Kettering cabinets have been in use in the Mayo Clinic, there has not been sufficient experience in treating diseases of the eye by this method to compare the therapeutic effect with that of fever produced by foreign proteins.

Interstitial keratitis in association with hereditary syphilis is seen more often in persons with the juvenile type but also occurs in those with the adult form in the third and fourth decades of life, particularly in women. Regardless of when treatment is begun and how strenuously it is applied, the disease, once established in one eye, affects the other similarly. The treatment of interstitial keratitis is the same as that of syphilis, with the addition of the employment of mydriatics. The use of the heavy metals, such as mercury, bismuth, arsenic, potassium, silver and antimony, in some form has for years been considered the basis from which all other forms of treatment have been evolved. The introduction of fever therapy has not supplanted the use of heavy metals but has materially added to their effectiveness. The duration of photophobia in interstitial keratitis is shortened by half, and the infiltration of the cornea is less extensive when, in addition to chemotherapy, induced fever is utilized. The treatment is much more effective in the first attack than in recurrences.

Other inflammatory syphilitic diseases of the eye, particularly uveitis, respond as well as interstitial keratitis. The response of patients to treatment varies so much that the intensity of the treatment is varied by the syphilologist to meet the needs in the case. Fever therapy alone is admittedly not as effective as when it is combined with the administration of heavy metals, but when it is added to classic medical treatment it materially increases the effectiveness of the latter.

Chemotherapy may be made more effective through the induction of fever by hot baths, vaccine, diathermy or air-conditioned cabinets. The means of inducing fever must be determined by the condition of the patient. Experience with the Kettering cabinet has shown that a temperature of 105 F. may be maintained for five hours with little risk to the patient if proper precautions are used. This method of applying heat, however, is expensive, and its application is necessarily limited. I agree with the authors that the best effects are to be obtained in diseases of the eye that are accompanied by cellular infiltration, such as uveitis, cellulitis and keratitis, and that induced fever is an adjuvant to and not a substitute for arsphenamine, bismarsen and other preparations of heavy metals.

Dr. Elmer L. Whitney, Detroit: Dr. Culler and Dr. Simpson have been modest in avoiding any discussion of the history and development of the Kettering hypertherm. One has to see and use this apparatus to appreciate fully the advancement that has been made by it in artificial fever therapy. The remarkable control of the height and duration of the fever and the easy access to the patient for observation during the treatment are gratifying.

My associates and I have followed the methods of treatment as described by the authors of this paper. Fifty patients with ocular disease have received fever therapy. Practically all these persons have been treated for conditions other than syphilis, and the results have given us encouragement and a desire to continue the treatment. We have treated ulcer of the cornea, keratitis, iritis and other intra-ocular infections produced by penetrating wounds.

Many patients object to the treatment with the hypertherm, especially private patients, and some refuse to continue. This depends on the discomfort the patient will undergo for relief of his ailment. Sufficient

sedation during the treatment is important. The treatment is not without danger. Two patients collapsed while in the cabinet, seemingly owing to cardiac failure. Children tolerate fever therapy well. The youngest child treated was 4½ years old. However, we have had difficulty in raising the temperature to the desired level in some children. I should appreciate Dr. Culler's comment on this point. The economic value of this form of fever therapy to the patient or to the county or the state is important. As compared with the time required for hospitalization during malarial treatment, the period needed for this form of therapy is reduced by two-thirds. The patient is able during treatment to continue his occupation.

Ocular syphilis is probably the disease best suited to artificial fever therapy. Every physician is aware of the inadequacy of chemical treatment in many instances. The reaction of syphilis in the eye is largely toxic. Exudates that are produced are most effectively removed by artificial fever.

Dr. Culler's results are striking and lend encouragement to further work. We have observed only 2 patients, and in spite of the poor results obtained, we believe that fever therapy is definitely indicated in the treatment of ocular syphilis.

Dr. Arthur M. Culler, Dayton, Ohio: As Dr. Benedict has indicated, the use of humidified hot air to produce fever is preferable because it is constantly under control. It also minimizes the debilitation of the patient which follows production of fever by the injection of typhoid vaccine and other methods.

As Dr. Whitney has pointed out, a sound vascular system is necessary as a prerequisite for the treatment. In regard to fever therapy in children, we have encountered no difficulties in maintaining the temperature at the desired level. While we regard this work as still in the experimental phase, the results are sufficiently promising to encourage others to carry out similar investigations. The production of artificial fever with temperatures at a high level requires special training of physicians and nurses. None of our patients was injured by the treatments.

ASSOCIATION OF ECTOPIA LENTIS WITH ARACHNODACTYLY

FRANK E. BURCH, M.D.
ST. PAUL

Marfan in 1896 described a peculiar syndrome, appearing usually in children, characterized especially by lengthening and thinning of the bones of the extremities. He considered the condition to be congenital or familial and suggested for it the name dolichostenomelia. In 1902 Mery and Babonneix reexamined Marfan's patient and considered the general defects due to failure of normal development of the epiphyses. In the same year Achard described a case of this syndrome in which the hereditary or familial features were marked, and because of the peculiar long, thin and tapered, clawlike finger-ends he suggested the present name arachnodactyly ("spider digits"). This name still persists though many variations of this syndrome with various accompaniments have been observed and the term is no longer entirely descriptive. Pediatricians and orthopedists chiefly were interested, as most of the earlier cases were in children. Ormond, in 1924, first drew particular attention to the ocular abnormality which frequently occurs in association with arachnodactyly. This feature has made the condition of great interest and attraction to oculists, since it is found that the chief ocular symptoms (ectopia lentis, iridodonesis and myosis) are observed in about half the cases.

At the present time there is a fairly large bibliography of foreign ophthalmologic publications on the subject, although until recently ophthalmologists and ophthalmologic societies have not given much heed to it. In fact, one learns on inquiry that comparatively few ophthalmologists in America have recognized the peculiar skeletal changes, the muscular dystrophy or the cardiac involvement which frequently is associated with congenital ectopia lentis. The American literature is remarkably scanty; Park and Powers in 1920, Piper and Irvine-Jones in 1926 and Patterson, of Montreal, in 1933 reported cases of arachnodactyly, but no particular attention was given to the ocular complications. Comparatively few abstracts of foreign articles are to be found in English journals, and these for the most part have appeared

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Read before the Section on Ophthalmology at the Joint Session of the American Medical Association and the Canadian Medical Association, Atlantic City, N. J., June 12, 1935.

during the past few years. In July 1934 Dr. Ralph I. Lloyd, of Brooklyn, read a paper before the American Ophthalmological Society in which he called attention to the ocular changes, and in the same month, at the Denver postgraduate summer course, I presented an account of the association of ectopia lentis and arachnodactyly. My attention was first called to the subject by Dr. Benjamin J. Dvorak and by Dr. Wallace H. Cole, who have kindly permitted me to incorporate here the histories of the patients they have observed in the orthopedic clinic. (One of these patients [case 2] was not actually seen by me, but the case is included in my reports.)

I have considered the entire subject of sufficient interest to American oculists to be reviewed and summarized, particularly from the standpoint of the relationship of arachnodactyly to ectopia lentis and other ocular changes. A description of a series of new case reports which I have collected and of a group of patients whom I have personally observed is contributed. An attempt has been made to explain the etiology of this condition and, finally, to furnish a bibliography of all important references, which will be of assistance to those who may wish to study the detailed published reports.

It cannot be stated that ectopia lentis and arachnodactyly are dependent on each other; ectopia lentis, of course, occurs independently of, and is associated with, a number of conditions other than so-called arachnodactyly. All that can be asserted here is that ectopia lentis is probably present in at least half, and probably more, of the cases of arachnodactyly and analagous anomalies of growth and that the etiologic factor or factors responsible for arachnodactyly are also involved in some way with the causation of ectopia lentis.

SKELETAL DEFFECTS

The commonest clinically observed abnormalities in the syndrome of arachnodactyly are in the long bones, joints, muscles, heart and eyes. While the skeletal changes predominate, cardiac lesions are commonly present, and other visceral abnormalities or peculiarities elsewhere may occur. The appearance of arachnodactylic children is absolutely typical. They are awkward-looking, being "all arms and legs." and are usually thin, with excessively long, slender limbs. The face is thin, often wrinkled and old-looking, and sometimes has a pained expression. Frequently there are changes in the spinal column. The entire skeletal system is generally affected, but the principal changes are found in the long bones, in the spinal column and in the extremities. While not often observed at birth, usually by the third or fourth year these skeletal changes become prominent when taken into account with the age, and there is a great disproportion when the length of the extremities is compared with the height. These changes are definitely established

by the tenth year. There is not only a real increase in the length of the extremities as compared with that of children of the same age but also a relative increase in comparison with the height. Attention is usually drawn to the abnormal elongation of the feet and hands, with the unusual thinning and tapering of the metacarpal and metatarsal bones. The delicate, spidery fingers are sufficiently characteristic to give the condition its name.

It is nearly always true that despite the tallness, the weight is below normal. The height of the patient reported on by Ormond and Williams was 6½ inches (16.5 cm.) above the normal height for his age, and the weight was 18 pounds (8 Kg.) below the corresponding normal weight. In the four cases reported by Young exact measurements were recorded, and roentgenograms were made of every phalanx and metacarpal bone in each case. These were compared, respectively, with similar measurements from roentgenograms of normal children of corresponding ages. Young found that in the arachnodactylic children there was a definite increase in the length in almost every measurement. In one of his patients the figures obtained at the age of 7½ years were for the most part equal to or greater than those for a normal child at the age of 14. The results proved that the phalanges and metacarpal bones exhibit in arachnodactyly a type of partial gigantism. Young's figures showed also that the greatest relative increase in length was in the terminal phalanx and that the ratio of the sum of the lengths of the phalanges and metacarpals of the digits to the total body length was higher in the arachnodactylic than in normal children. The same condition is found in the feet as in the hands, but, owing to more concomitant deformities in the feet, it is extremely difficult to obtain accurate roentgenograms. Flatfoot, often with calcaneal vertical spurs, is common, and the great toes are often badly deformed, as in my cases 2, 3, 4 and 6. The skeletal changes differ from the gigantism of acromegaly, in which the thickness as well as the length of the bones is increased. In arachnodactyly roentgenograms show no true pathologic changes in structure, the bones being simply elongated.

Abnormality of the skull is common in cases of arachnodactyly, and, according to Young, in 65 per cent of cases the dolichocephalic variety is present, although in a few cases, as in those of Thaden and of Kallius, the skull has been reported to have been smaller in relation to the general size. Other cranial deformities that are often noted clinically are predominant supra-orbital ridges, bossing of the frontal eminences, a prominent or broad and sunken nose, depression of the base of the skull, a prominent chin and a narrow arched palate, occasionally cleft, as in a case reported by Piper and Irvine-Jones and another by Zuber. The face is generally long and narrow, and the skin is wrinkled. The external ears are often malformed and abnormally

curved, with enlargement of the lobe, as in the cases of Börger, Fowler, Poynton, Pfaundler, Salle, Thomas and others. Thomas described the deformity of the ear as follows: "Auricles protrude away from the head in their upper half and cling in their lower half. The anthelix and crus helicis are usually more developed than the helix. The auricular cartilage in general is poorly developed." Dentition is usually normal, but in a few cases double rows of teeth have been noted, and the teeth may be long and narrow like the bones. Thoracic deformities are found in 50 per cent of the cases, according to Young, the funnel-shaped variety being most frequent. Pigeon breast is also seen often, and in some cases there is projection of the lower portion of the sternum or flattening of the whole or part of the chest wall. Thoracic deformities, mostly flatness and narrowness of the chest, were noted in four of my own cases (cases 3, 5, 7 and 8). *Trichterbrust* was present in cases 1, 2, 4 and 5. The scapulae have been described as winged or protruding in a few cases; this deformity has been especially mentioned by Börger and by Ormond. I did not observe it except as a result of spinal changes.

Thaden has said that some degree of spinal deformity exists in almost all cases. A large number of the reports of cases of arachnodactyly have mentioned this condition, which apparently is not of itself a congenital deformity. It probably develops later from the congenital defects, as a result of general weakness of the vertebral ligaments and poor muscular development but perhaps also because of the slenderness of the bones. Kyphosis, especially of the dorsal region, is observed either alone or combined with compensatory lordosis or scoliosis, that is, kyphoscoliosis. This was observed in several of my cases. Spina bifida occulta has been observed several times: in two of the familial cases of Lloyd and in three cases reported by Brock, Zuber and Weill, respectively. Exceptionally, there is hypertrophy of the spinal apophyses.

The joints often show marked looseness, especially those of the elbows, knees and fingers, with laxity of the ligaments. In my case 3 there was unusual mobility of the patella. In case 4 there was extreme weakness of the ankle joints, and in case 5 there was abnormal flexion of the elbows. Weill and others have mentioned a marked retraction of the flexor tendons, especially of the joints of the fingers and toes, which prevented complete opening and closing. Other patients showed extensive contracture of the joints, with inability to supinate fully. In the case reported by Poynton, a congenital flexion of the thighs was so pronounced that the child resembled a chimpanzee. This is suggested in one arachnodactylic patient personally observed (fig. 1) in whom, however, there was no ectopia. Börger, Killmann, Schreiber and others have referred to the hyperlaxity of the distal joints, which at times, however, may be associated with ankylosis of the proximal joint.

DEFECTS IN SOFT TISSUES AND VISCERA

Muscular tissue is generally poorly developed, yet ordinarily the strength is in proportion to the muscular girth. The muscles of the trunk are generally affected, yet there is generalized muscular dystrophy, which to some observers suggests a resemblance to congenital amyotonia. It seems probable that the growth of the muscles and tendons does *not* keep pace with that of the bones. Poor generalized muscular development was noted in cases 2, 4, 5, 6 and 8.

Fig. 1.—A typical arachnodactylic child, showing a chimpanzee-like posture, spinal deformity, pigeon breast, clawlike fingers, abnormal feet, absence of adipose tissue and poor muscular development. The eyes are normal.

Scarcely any subcutaneous fat is present, which explains the emaciated appearance of some patients. Many of the reports state that in some instances arachnodactylic children are of normal weight and are plump at birth, later seeming to lose the fatty tissue. In a few special cases the adipose tissue was normal, as noted by Carrau and by Kallius. Adipose tissue was notably deficient in my cases 2, 4 and 8.

The heart is more or less seriously affected in a large proportion of these patients, as especially noted by Viallefont and Temple. Weve said that about 30 per cent of patients show this; Killmann gave the figure as 40 per cent; but Thaden said that there is a systolic murmur in 60 per cent of the cases. Auscultation usually discloses a systolic or presystolic murmur. The causes are incompetent valves, a patent foramen ovale, as in Pfaundler's case, or some other congenital defect of the heart. A tubular or displaced heart is not unusual and in some instances is attributable to thoracic deformities. Piper and Irvine-Jones considered congenital cardiac disease a prominent feature of arachnodactyly, and it is recorded in the autopsy report of their patient. Deformities of blood vessels, for instance of the abdominal aorta, have been described. The blood pressure is usually low, and the pulse rate is slow. In my series the cardiac symptoms were pronounced in four instances (cases 2, 6, 7 and 8). One patient (case 2) died suddenly of some cardiac disorder.

Pulmonary changes have occasionally been observed. In one of Börger's cases there was a defect of the middle lobe of the right lung. Piper and Irvine-Jones noted a small middle lobe in the right lung, and the left lung consisted of one lobe only. Pfaundler's patient had only two lobes in the right lung. Deformities of the thorax easily give rise to pulmonary complication and pneumonia. The nervous system and the mentality are not affected, as a general rule. The general intelligence is often especially good and consistent with the possibilities of education. Moro has drawn attention to a neurologic type of arachnodactyly. The blood picture is usually normal.

OCULAR DEFECTS

The ocular complications are those associated with ectopia lentis-iridodonesis, small, contracted pupils, which fail to dilate normally under the influence of atropine, and anterior chambers of uneven depth. Occasionally nystagmus or divergent squint is present. Lenticular myopia is usually found if the lens is still behind the pupil, although axial myopia also may occur. The myopia in some instances has been reported as high as from 40 to 60 diopters. Hypermetropia, generally of low degree, is less common. It is usually present if the ectopia is sufficient to simulate aphakia, as in my cases 1, 5 and 6. A persistent pupillary membrane, with clouding of the anterior capsule, was noted in one of Börger's cases; coloboma of the lens was noted in Franceschetti's case, in King's case, in one of Weill's, in one of Weve's and in others. Paralysis of accommodation was reported by Weve. Ormond noted abnormalities of the retinal vessels, and one of Weve's patients

showed also chorioretinitis. Accompanying edema of the lids was observed by Salle; Piper and Irvine-Jones' patient showed shortness of the lower lids.

Ocular symptoms were noted by Ormond in eighteen of the thirty-four case reports that he collected. Weve, in 1931, after a thorough study of the literature, found reports of eighty-four cases of arachnodactyly, twenty-three of which were new cases observed by him or by his colleagues. He stated that ocular complications were present in 50 per cent; and in 33 per cent of the eighty-four cases, including ten of his own twenty-three cases, there was ectopia lentis. These figures would probably be much higher if a more careful examination for ocular symptoms had been made in the earlier reported cases of arachnodactyly; in cases reported since Weve's article was published the proportion of ectopia lentis has been much higher. Weill, in 1932, reported that within the previous two years he had seen eight patients with ectopia, six of whom presented the syndrome of arachnodactyly. Thaden stated that subluxation of the lens occurred in nine of twelve patients with arachnodactyly showing ocular symptoms, and Lloyd stated that five of the six patients seen by him showed it. Killmann reported seven cases of manifest arachnodactyly, in all of which there was congenital bilateral dislocation of the lens. Hudson reported the association in four members of one family. Cases have also been reported by Coppez, Kurz, Weber, Vogt, Padovani, Viallefont and Temple, Christophersen, Amsler and probably others. These with the series personally reported bring the total number of cases reported in which there existed an association of arachnodactyly with ectopia lentis to over seventy.

Ectopia lentis and other malformations of course occur in association with several other anomalies of growth besides arachnodactyly. A comprehensive review by Ringelhan and Elschnig should be consulted for full details of clinical and other aspects of this malformation. It may be stated positively that ectopia lentis has been found in at least one third of the arachnodactylic persons observed and that it is always congenital. Ringelhan and Elschnig gave as their opinion that ectopia lentis is a developmental defect, the causes of which are anomalies in the anterior bulbus (75 per cent of cases) and a myopic elongation of the eye.

Bilateral dislocation of the lens is the most common and characteristic ocular finding. The dislocation is often incomplete; and although the ectopia is mostly upward, it has been observed in all positions, frequently being downward or lateral. This was true in my cases 1 and 6. Sometimes the dislocation is complete, as in the cases reported by Thaden and Weve; frequently it is into the anterior chamber, and glaucomatous phenomena may be observed. The lenses are frequently of reduced

dimensions and may be opaque or calcified. Although ectopia lentis associated with arachnodactyly is congenital, it may not be observed until a long time after birth.

HEREDITARY AND FAMILIAL TENDENCIES

A hereditary, or at least a familial, origin is definite, both for ectopia lentis and for arachnodactyly, in view of the reports of de Haas, Weve, Lloyd and several others and of two families in my group. In the reports by de Haas the two conditions were present in a brother and sister. Weve reported the cases of a father, two sons and a daughter, all with arachnodactyly and ectopia lentis; a grandfather also apparently had arachnodactyly. Thaden said that in general in cases of ectopia lentis the frequency of heredity is relatively great, although it was not present in his own patient associated with arachnodactyly. Hereditary evidence was observed in two of Weill's cases; Kurz reported the presence of ectopia lentis associated with arachnodactyly in two sisters but could find no hereditary history. In five of Killmann's seven cases there was evidence of familial or hereditary transmission. Viallefont and Temple reported on a family in which the father and three of the children showed ectopia lentis and the three children were arachnodactylic. The hereditary factor could not be traced beyond the father. Lloyd's description of a family of eight in which four children showed arachnodactyly with bilateral dislocation of the lens is interesting. The father was normal. The mother had the skeletal features of arachnodactyly but normal eyes. One of my family groups is similar to Lloyd's. Available details of cases reported seem definitely to point to inheritance on the maternal side of the family.

Of course ectopia lentis also may be hereditary or familial, with no evidence of Marfan's syndrome. Kredbova's report illustrates this. Throughout four generations there were thirty-seven relatives with a familial condition, fifteen with ectopia, two with congenital colobomas and six with myopic changes in the fundus; but none of his cases was a typical case of arachnodactyly. Weill reported on two cases of ectopia in adult dwarf females who were not arachnodactylic but one of whom had an arachnodactylic child. Other anomalies, such as those of the external ears, palate and nails, may be associated with ectopia lentis independently of other evidences of arachnodactyly.

ATYPICAL FORMS

At this point mention should be made also of what the French clinicians term the formes frustes, really disguised or masked forms in which the manifestations of arachnodactyly in familial or hereditary histories are revealed in connection with ectopia lentis. Apparently the more frequent ocular manifestations of these forms are megalocornea,

as reported by Fleischer and by Thaden, and megalophthalmos with or without dislocation of the lens. I observed spina bifida in two sisters, and other spinal deformities were marked in one. These two sisters are not included in my series, their arachnodactyly being of the fruste type. One had megalocornea and ectopia; the other, true buphthalmos, with ectopia lentis developing during childhood, and subsequent glaucoma. These sisters showed also marked contractions, deformities of the feet and cardiac changes. A third patient with megalocornea and ectopia lentis associated with peculiar contractures of the elbows and fingers and with spina bifida occulta was observed by me at the University of Minnesota Hospital, classified in the orthopedic clinic as having a fruste type of arachnodactyly. These three patients all had megalocornea or buphthalmos; in two glaucoma developed, requiring operation. In the third patient, both lenses were in the vitreous, the sight of one eye being lost, with detachment of the retina, after attempted removal of the luxated lens, the other still retaining vision of 20/40 with correction. In the forme fruste associated with ectopia lentis one may fail to find the usual characteristic symptoms of arachnodactyly, such as the spider digits, muscular dystrophies or marked elongation of the extremities. In reality, only if typical changes are found and there is a familial case of arachnodactyly is one justified in classing the patient as arachnodactylic. In one of the familial cases reported by Weve all the bulbi were enlarged, but not the corneas. The explanation of such a case has been given by Igersheimer, Thaden and others as a small diameter of the lens, which they considered to be fairly common in arachnodactyly. Igersheimer also explained megalocornea as due to an inequality in the rapidity of growth of the lenses and of their investing membranes, leading to tension of the zonular fibers. In cases in which the lens subluxated into the anterior chamber it was found to be very small, this finding being confirmed by slit-lamp examination. The smallness may be due in part or entirely to marked convexity of the lenses, to poor nutrition or to insufficient embryonic vascularization. In one case Weill noted microphthalmia.

Weber remarked:

> There are doubtless congenital developmental conditions allied to arachnodactyly, in some of which the spider finger feature may be absent. One may find the limb feature, especially the upper, as the chief or only characteristic in some cases. In others, the spinal deformity, the heart lesion, or muscular dystrophy may be outstanding.

Weber reported the cases of a brother and sister, both of the asthenic type, with bilateral congenital dislocation of the lens, who were the youngest of a family of fourteen. The parents seemed normal, and there was no hereditary history.

SEX AND AGE

As regards sex incidence, Weve found forty cases in females and forty-two in males in the eighty-four reports which he collected in which the sex was stated. Since then cases have been reported on by Vogt, Ingram and Inglis, Hudson, Greyer, Pentagna, Weill, Killmann, Lloyd and a number of others. Several of these have reported new cases. Weill reported on eight cases; Lloyd, six, and Thaden, twenty-three. So at the present time the total number of cases reported in the literature is in the neighborhood of one hundred and twenty. It is probable that arachnodactyly occurs much more frequently than is believed. In the observation of congenital ectopia lentis during recent years, ophthalmologists have looked for arachnodactyly with assiduity since the association of the two conditions has been brought to their attention, as signified by the more frequent reports in the literature recently. Now that the attention of ophthalmologists and others in this country has been drawn to the subject, the total number of reports of cases may be greatly increased. Although undoubtedly the abnormality exists from early life, it seems to have been rarely observed at birth. In none of my patients was any peculiar change noted at birth. In the case reported by Ormond and Williams the condition did not manifest itself until the child was 4 years of age. In the case reported by Poynton and Maurice the patient was 14 when first observed. Weve reported the case of a patient aged 40 and that of another aged 62. In several other reports the condition was first brought to prominent attention in adult life.

REPORT OF CASES

CASE 1.—Alice L., aged 8 years, was admitted to the University of Minnesota Hospital on Aug. 18, 1919, with a diagnosis of subluxated lenses and an anomaly of growth. The family history did not reveal any physical abnormalities or ocular defects. The child's birth was normal at term; she was the fourth of eight children and was considered physically perfect at birth. As an infant she would lie quietly for hours, apparently not noticing anything, and only when she was 2 years old was it definitely determined that both eyes were extremely near-sighted. She walked at 18 months and talked when 2 years old. At about that time it was noticed that she was growing rapidly. At 5 her slim body, long arms and legs and weak ankles were frequently commented on. She had measles, pertussis and influenza, without special sequelae, and aside from her rapid growth and defective eyesight she was regarded as a healthy child. When admitted to the hospital she was a diphtheria carrier and had enlarged infected tonsils and marked involvement of the cervical lymph glands. There was a funnel-shaped depression of the chest, the left border of the heart being in the midaxillary line. A systolic murmur was noted at the apex and also over the precordium; the pulmonic second sound was greatly accentuated, being heard best in the second interspace. The extremities were very long and thin, with long tapering fingers and toes. There were slight kyphosis with compensatory lordosis and scoliosis with convexity to the left in the lower portion of the dorsal region. A complete absence of fatty tissue was

noted. The basal metabolic rate was 17 and 20 per cent below normal, respectively, on two tests. The Mantoux and Wassermann reactions were negative. The blood count showed: hemoglobin, 74 per cent; erythrocytes, 4,464,000; leukocytes, 9,400; polymorphonuclears, 50 per cent; lymphocytes, 46 per cent; large mononuclears, 3 per cent, and eosinophils, 1 per cent. The sugar tolerance test was normal on two occasions. Roentgenographically the sella turcica was observed to be small but within normal limits. Tonsillectomy was performed successfully with the patient under ether anesthesia.

Examination of the eyes revealed that each lens was subluxated *downward*. The patient was able to count fingers at 1 meter. She could read large print with either eye. The vision could not be improved with lenses at that time. On April 13, 1934, I reexamined the vision. In the right eye it was 2/200; with +5.50 sph. ⊂ +1.25 cyl., axis 120°, 10/200. In the left eye it was 10/200; with +3.50 sph.,

Fig. 2 (case 1).—Spinal deformity accompanied with *Trichterbrust*. The heart extends to the left midaxillary line. This young woman is 6 feet, 2 inches (188 cm.) tall and wears 11-4A shoes. The extremities are long. There is ectopia downward. With aphakic correction the vision in the right eye is 10/200; that in the left eye, 20/200.

20/200. Without addition she read Jaeger's test type 0.75 at 2 inches (5 cm.) with each eye; with +3.00 sph. added both right and left she read Jaeger test type 1.50 at 6 inches (15 cm.). There was a distinct absence of orbital fat, and the eyes were deeply set. There was no nystagmus; the left eye was slightly exotropic. The pupils contracted equally, measuring 2 mm. in diameter; after atropine had been administered they measured 5 mm. The lenses were displaced downward and outward, but they fell upward when the face was down, so that they covered the pupillary space. They were distinctly and uniformly opaque. The tension in each eye was 20 mm. (Schiötz-Gradle). The light blue irides had an abnormal pattern that was distinctly radial. Iridodonesis was marked.

At an examination made by Dr. Benjamin Dvorak on May 25, 1933, the following note was made: "The patient seemed mentally normal. The arms extended almost to the knees, the hands showing distinctly arachnodactylic characteristics. The feet were long, size 11-4A shoes being worn. The patient was very tall and slight, with long arms, long legs, very long fingers and long feet. The head had a somewhat long anteroposteror diameter, with a high palate and narrow mandibles. There was no noticeable deformity of the spine, but there was a marked funnel-shaped deformity of the lower portion of the sternum, the depression showing a depth of 6 cm. The photographs and roentgenograms showed that there had been little change from the general characteristics noted fifteen years previously."

The roentgenographic report was made by Dr. Leo Rigler. The bones of the hands and feet were unusually long, but there were no other evidences of abnormality. The carpals, radius, ulna and humerus appeared normal. The femurs, tibias, fibulas and ankles appeared normal except for an unusually increased

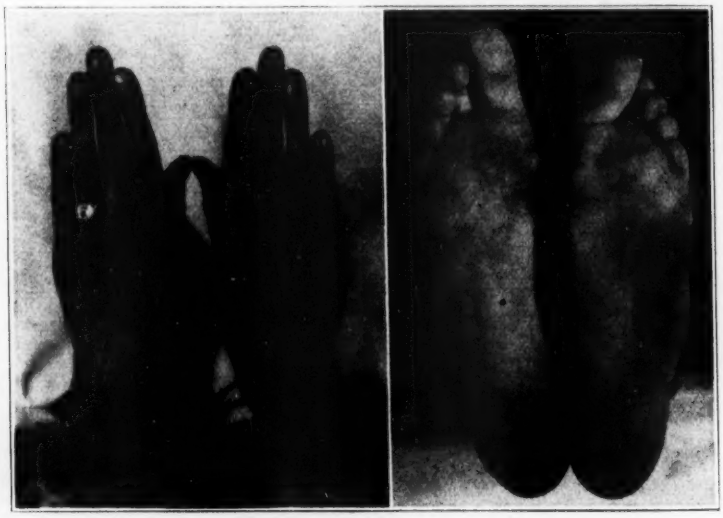

Fig. 3.—Hands and feet of the patient shown in figure 2.

length. There was some bowing of the middle portion of the tibia and of the fibula. There were also an unusual number of growth lines in the distal end of the tibia. The shoulders, scapulae and clavicles appeared normal. The ribs were normal except for an unusual sloping. The knees were normal. There was marked scoliosis in the thoracic region of the spine, with considerable axial rotation, and scoliosis in the cervical region was also present. Marked lordosis in the lumbar region of the spine and some scoliosis and axial rotation were present. The fifth lumbar vertebra was unusually deep. There was marked deformity of the sternum, with an extreme degree of displacement posteriorly, the appearance being characteristic of *Trichterbrust*. The skull appeared normal. The heart showed displacement well to the left side of the chest. This may have been secondary to the deformity of the chest and the scoliosis. Considerable rotation of the heart and an apparent dilatation of the right ventricle were noted. These may have been associated with a congenital cardiac lesion, but it is more likely that they were due to the displacement of the heart than to any intrinsic lesion. There was a suggestion of scalloping of the ribs, such as might occur with coarctation of the

aorta, but the arch of the aorta and the descending portion appeared to be normal. The possibility that there was an unusual collateral circulation through the intercostal arteries must be borne in mind, however.

At an examination by Dr. A. R. Hall, of the staff of the university, on Feb. 18, 1935, the following report was made: "The heart was markedly displaced to the left by the deformity. The total transverse diameter of the heart was 12.5 cm. The apex beat could be felt in the sixth and seventh interspaces, 13 cm. to the left of the midsternal line. The heart beat was regular, and the pulse rate was 84. A short, soft systolic murmur was heard in the pulmonary area that was not conducted

Fig. 4 (case 2).—G. W., aged 10, showed evidences of serious cardiac involvement and died suddenly at the age of 11. Note the peculiar limbs and the absence of adipose tissue.

to any appreciable distance. The cause could not be definitely determined, but the murmur probably belonged to the group of so-called functional murmurs. The blood pressure was 102 systolic and 70 diastolic."

Summary.—This patient (figs. 2 and 3) showed the characteristic mesoblastic growth anomaly of the gigantism type associated with congenital ectopia lentis.

CASE 2.—Glen W., aged 10, was admitted to the service of Dr. Wallace Cole in the Shriners' Hospital on Sept. 20, 1927, with a diagnosis of residual infantile paralysis involving the lower limbs. The family history revealed nothing per-

tinent. He had been delivered instrumentally at full term. Considerable deformity of both lower limbs was noted at birth. He had a doubtful infantile paralysis at 3 years of age, chickenpox at 5 and measles at 9. He did not talk until he was 3. When he was 4 years old he was examined by the late Dr. Harold Gifford and Dr. James Patton, who found ectopia lentis, miosis and iridodonesis. The ectopia lentis was probably incomplete, as on admission he was wearing —8.00 sph. lenses. There was no record of the vision but merely a notation that there were myopia of 10 diopters in each eye, miotic pupils, iridodonesis and subluxation of both lenses, their position being undetermined.

The child's appearance was impressive, as his head was of unusual contour, with a high forehead, prominence of the occipital bone and a very high, narrow dental arch. He was abnormally tall for his age, with a long, narrow, flat chest, limbs of unusual length and a poorly developed musculature. The feet also were exceedingly long and narrow, with elongation of the great toes, and he wore size 10½-4A shoes. The absence of adipose tissue, the dry skin, the visible pulsation of the carotid arteries and the poorly developed musculature suggested some endocrine dysfunction.

Fig. 5.—Roentgenograms of the feet of the patient shown in figure 4.

The physical examination revealed no deformities indicative of chronic changes. The thyroid gland was normal. Examination of the thorax showed evidence of poor nutrition, with retraction of the intercostal space and with definite enlargement of the costochondral joints. The lungs were normal. Examination of the heart showed general precordial pulsation, a systolic thrill at the apex and definite cardiac hypertrophy, but no arrhythmia. A high pitched mitral murmur, with reduplication of the pulmonary second count and a double murmur heard at the apex, and a diastolic whistling murmur also were noted. The systolic murmur increased toward the base. The pulse rate while the patient was resting was 80; after he walked to the end of the room and back it was 102, with the murmur increased and the precordial pulsation markedly heaving.

Neurologic examination showed that the ankle and knee jerks were absent on the right side; on the left they were sluggish. No ankle or patellar clonus, no pathologic toe signs and no marked sensory changes were noted. The neurologic impression was one of primary myopathy. The child was mentally bright.

The laboratory tests revealed no abnormalities. The Wassermann reaction was negative.

The knees were hyperextended, with luxation of the sternoclavicular joints, especially on the right. Both feet showed marked valgus deformity with elonga-

tion, especially of the great toes. The deformity of the chest was attributed to rickets and cardiac changes. Dr. Cole diagnosed the condition as congenital arachnodactyly.

Summary.—This was a typical case of arachnodactyly with a marked ocular syndrome and serious cardiac involvement. The deformity of the lower limbs was noted at birth. Death occurred suddenly as a result of cardiac disease within a year after the patient left the hospital.

CASE 3.—Frederick S., aged 12, was first seen by me at the State School for the Blind on April 12, 1933, when his peculiar appearance and abnormal extremities were noted. On April 4, 1935, he was admitted to the Miller Hospital, in St. Paul, for observation. An attempt to improve his vision with glasses when he was 7 had

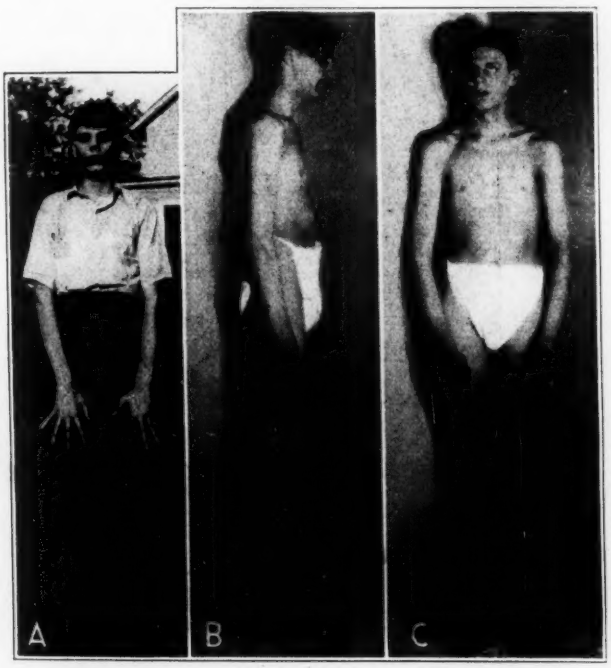

Fig. 6 (case 3).—*A*, F. S. at the age of 12; *B* and *C*, at the age of 14. He is 6 feet, 2½ inches (189 cm.) tall, weighs 139 pounds (63 Kg.) and wears size 12 shoes. There is slight spinal curvature. The heart is hypertrophied. There is ectopia upward. In the left eye retinal detachment is present. The boy has typical spider digits.

been ineffectual. The family history revealed no abnormal physical characteristics or ocular defects on either side of the family. This boy was an only child. He weighed 10 pounds (4.5 Kg.) at birth. He had always been taller and thinner than other children of his age. This was first noticed in early childhood.

Dr. Henry Wagener, of the Mayo Clinic, reported that the boy was first examined on Aug. 24, 1931. Vision in the right eye was 6/60; with the left eye only moving objects were noted. Each lens was opaque and luxated upward and backward. In the left eye there was a large detachment of the retina. An operation for removal of the luxated lens of the right eye resulted in loss of vitreous.

Examination on April 4, 1935, showed phthisis bulbi of the right eye, minus tension and evidences of previous iridocyclitis. The left eye was apparently externally normal. The pupil was miotic; it contracted to about 3 mm. and reacted to light. Iridodonesis and a deep anterior chamber in its lower half were noted. Several instillations of epinephrine hydrochloride and atropine dilated the pupil to about 7 mm. The lens was dislocated almost directly upward and was uniformly opaque. The retina was detached through its lower half. The upper portion of the fundus could not be seen. With the pupils dilated fingers were counted at 8 inches (20 cm.). No treatment was ordered.

Physical and orthopedic examinations by Dr. Harvey Beek and Dr. George Williamson revealed the following: The boy was abnormally tall for his age; he was "all legs and arms," with long limbs, large feet and spidery fingers, and he had a postural defect. He was 74½ inches (189 cm.) tall and weighed 139

Fig. 7 (case 3).—A roentgenogram of one hand superimposed on that of an average adult 6 feet, 2 inches (188 cm.) tall.

pounds (63 Kg.). The complexion was sallow. The hair was very coarse, the skin was dry and there was deficiency of adipose tissue. The musculature was poorly developed but showed no amyotonia or paresis. The chest was broad and flat, with a large sunken area over the xiphoid process. There was no evidence of rickets. The heart was enlarged to the left in the third and fourth interspaces in the region of the left auricle. There was a sinus arrhythmia, but no murmurs were present. The blood pressure was 110 systolic and 70 diastolic. The pulse rate was 80. The thyroid gland appeared to be normal. The basal metabolic rate was minus 8. The laboratory examinations revealed no abnormalities.

Further orthopedic examination revealed that the skull was normal in all respects. The physiologic dorsal and lumbar curves were markedly exaggerated. All the joints showed moderate relaxation with a resulting increase in the range of motion. There were no flexion deformities or other abnormalities. There was a

marked relaxation of the structures in which the patella is developed, so that it was possible to dislocate the patella in either condyle of the femur. The extremities, although thin, were symmetrically developed throughout. No atrophy was apparent on either side.

Examination of the neuromusculature system showed that all the tendon reflexes were normally active. The hands and feet were normal except for the marked enlargement. The boy wore size 12 shoes.

Roentgenograms showed only slight scoliosis in the upper dorsal portion of the spine; the heart shadow was of the drop-heart type. No pathologic condition was noted in the bones of the feet or hands, only a general enlargement of all the bones.

Summary.—This boy was a lusty infant. Aside from the eyes, the arachnodactylic phenomena, other than the skeletal defects, were not prominent, only the abnormal length of the limbs, feet and hands being outstanding.

CASE 4.—Mary B., aged 25, was referred to me by Dr. H. J. Dvorak[1] for ocular examination on Nov. 7, 1933. An abstract of the early history of this patient was kindly furnished by Dr. Henry Wagener, of the Mayo Clinic. None

Body Measurements in Centimeters

Arms			Legs (from anterosuperior spine to medial malleolus)	
Right arm	88		Right leg	105.5
Left arm	89		Left leg	104.0
Head (occipital frontal measurements)	45			
Fingers (from web to tip)	Right	Left	Toes	Right	Left
Thumb	7.00	6.50	First	5.0	5.00
First	9.25	9.00	Second	7.0	7.25
Second	10.00	10.00	Third	6.0	6.00
Third	9.50	9.50	Fourth	5.0	5.00
Fourth	8.00	8.00	Fifth	4.0	4.00

of her family or relatives was near-sighted or had any evidences of growth anomalies. Her sister had normal vision, was of ordinary stature and was in good health.

The patient weighed 8 pounds (3,628 Gm.) at birth (instrumental delivery) and seemed to be entirely normal. As a child she held objects very close to the eyes, and when aged 3 years she was taken to the Mayo Clinic on account of strabismus. She was given —2.00 sph. lenses. When aged 4 she was again taken to the Mayo Clinic, on account of weak ankles and a tendency to eversion and toeing in of the feet, especially the right foot. At that time it was noted that the action of the heart was markedly irregular. The deformity of the feet increased during the following year, and there was a small bursa on the external malleolus of each foot. When examined at the clinic on Dec. 22, 1922, at the age of 14, the height was 5 feet, 9¼ inches (177 cm.), and she weighed 118 pounds (53.5 Kg.). According to the report filed at the Mayo Clinic, there were moderate varus, thickened bursae of the external malleoli, undeveloped leg muscles, weakness in the peroneal muscles and moderate genu valgum. The extremities and hands were very long, and the deltoid muscles were not well formed. The reflexes were active, and no fibrillary twitching was present. The condition suggested abiotrophy and the Charcot-Marie-Tooth syndrome.

1. Dr. Dvorak published a report of this case in 1932.

Ten years later, on Aug. 16, 1932, the height was 5 feet, 9½ inches (176.5 cm.), and the weight, 114 pounds (51.7 Kg.). The hands were long and slender, the metacarpals and metatarsals being abnormally long. There was general muscular atrophy (hypotonia). The palate had a high arch. The heart showed chronic adhesive pericarditis but no congenital lesion. It was difficult to exclude the presence of mitral endocarditis. Roentgenograms of the skull revealed no abnormality. The abnormal length of the extremities suggested dysfunction of the endocrine glands. Urinalysis gave negative results. The blood count and hemoglobin value were normal. The Wassermann reaction was negative. Several

Fig. 8 (case 4).—M. B., aged 26, is 5 feet, 10½ inches (179 cm.) tall and weighs 108 pounds (49 Kg.). The head is dolichocephalic. There are generalized muscular atrophy and an absence of adipose tissue. There are enophthalmos, extreme miosis and iridodonesis. The lenses are clear. Vision in the right eye is 10/200 and in the left eye, 8/200.

operations were performed on the feet, with improvement. Examination in the ophthalmologic department of the Mayo Clinic on Nov. 20, 1922, showed that the vision in each eye was 3/60 with a —14.00 sph. The patient could read Jaeger's test type 0.62. The pupils were small and irregular, and the reflexes were normal. The irides were tremulous and were atrophic. Both lenses were dislocated upward and backward. No details of the fundus were made out. The condition of the eyes was practically unchanged when I made an examination on Aug. 16, 1932.

When I first saw the patient (Nov. 7, 1933) her appearance was striking. She was abnormally tall and thin, with deep-set eyes. She was 5 feet, 10½ inches (179 cm.) tall and weighed 108 pounds (49 Kg.). There was a definite absence of orbital fat, the exophthalmometric measurements being 10.5 mm. on the right and 10 mm. on the left. Vision with correction was as follows: In the right eye, with —16.00 sph. ⌒ —2.00 cyl., axis 90°, it was 10/200. She read Jaeger's test type 0.37 at 3 inches (7.5 cm.). Vision in the left eye with —16.00 sph. ⌒ —2.00 cyl., axis 90°, was 8/200. Jaeger's test type 0.37 was read at 2 inches (5 cm.). Iridodonesis was marked, each pupil measuring 1.5 mm.; under the influence of

Fig. 9 (case 4).—Left lower extremity.

atropine later it was found that they did not dilate beyond 5 mm. The iris patterns were absent, the irides being distinctly atrophic, as shown by slit-lamp examination. The tension of each eye was 11 mm. (Schiötz-Gradle). No view of the fundus was obtainable. The anterior chambers were deeper in the lower portion, which is indicative of ectopia upward. Both lenses were transparent.

Physical examination, made by Dr. A. R. Hall on March 18, 1935, revealed no particular complaint. The appetite was good; there was no indigestion or constipation, and only slight dyspnea was noted on exertion. There were no pains in the joints. The patient slept well. Menstruation was regular.

The head was somewhat long and wedge-shaped. The mandibles were narrow, and the palate was high-arched. The spine showed only slight scoliosis in the

lower dorsal region. The thyroid gland was normal. The chest was flat and funnel-shaped. The lungs were normal. The heart was essentially normal. There was a short, soft systolic murmur at the apex, possibly from a slight mitral regurgitation. There was no evidence of pericardial involvement at that time. The urine was normal. The hemoglobin value, blood counts and differential counts were normal.

During my fifteen months' observation of the patient the condition of the eyes was unchanged. She enjoyed life, was of high mentality and had been able to go through high school with average grades. She complained of some shortness of breath, but that may have been due to an inability to exercise caused by the pes cavus.

Summary.—This was a typical case of arachnodactyly with ocular symptoms. There was almost no spinal deformity, the chief characteristics being the long extremities, weak joints and diminished body weight, with the usual ocular picture.

CASE 5.—Clarence C., aged 8, was admitted to the University Hospital on Jan. 10, 1933, because of asthenia, malnutrition, poor vision and divergent squint. Three other members of his family (the father, aged 41; one brother, aged 5, and one sister, aged 12 [fig. 10]) had ectopia lentis, the lenses in all cases being dislocated upward; all had defective vision and were wearing strong plus spherical lenses. The mother, aged 38, and one brother, aged 9, showed no ocular abnormalities and had normal vision. The father and one sister showed only the characteristic arachnodactylic trend. The 12 year old sister (fig. 10) showed unusually long bones, spider digits, lordosis in the lower dorsal region of the spine, scoliosis to the right and an elongated chest that was moderately flat in the anteroposterior diameter. There was a short, soft systolic murmur at the apex. The father showed rather long thin hands and was pigeon-breasted, but otherwise there were no marked physical abnormalities. The 5 year old brother also was very tall and slim, and his head was rather long and narrow.

The patient weighed 7½ pounds (3.5 Kg.) at birth, which was at term. His health and growth were apparently normal until rickets was noted in the second year. Under good hygienic and feeding conditions and antirachitic treatment he failed to improve or develop normally. He seemed to be a weakling lacking in normal strength. He had never attended school, because of defective vision, which was evident during his first year. He always held small objects at a distance of approximately 4 or 5 inches (10 to 12.7 cm.) in order to distinguish them. He was listless and expressionless and seemed mentally dull.

On admission physical examination showed that he was 51 inches (129.5 cm.) tall and weighed 52 pounds (23.6 Kg.). He was pale and appeared to be anemic. The hair was thin and coarse. His head was somewhat large for his size. One was impressed with his general apathy and weakness. The fontanels were normal. Roentgenograms of the skull showed no anomalies, although there was some increase in the diploic veins and vascular markings for his age. The sella turcica was normal. The teeth were irregular, carious and lacking in enamel. The chest was flattened and asymmetric, with a marked funnel-shaped depression of the lower sternum. A rachitic rosary was noted. Cardiac pulsation was visible in the third, fourth and fifth interspaces, without precordial bulging; the cardiac measurements were regarded as normal for the height and weight. There were no other abnormal cardiac findings. The pulse rate was 110, and the systolic blood pressure was 100. The abdomen was flaccid but normal. The genitalia were normal. Neurologic examination revealed no abnormality except the general asthenia and distinctly subnormal mentality. There was no scoliosis or other spinal deformity.

The Mantoux and Wassermann tests were negative. An examination of the blood on March 6, 1935, showed 69 per cent hemoglobin, 4,120,000 erythrocytes, 5,950 leukocytes, 69 per cent polymorphonuclears, 26 per cent lymphocytes and 5 per cent monocytes. Urinalysis showed albuminuria. The general musculature was poorly developed, and the muscle test showed poor tonus and asthenia, evidently a definite amyotonia. The fingers and toes were moderately elongated and narrowed. Roentgen examination showed a congenital flattening and deformity of the distal phalanges of the hands and feet. They were moderately arachnodactylic in type.

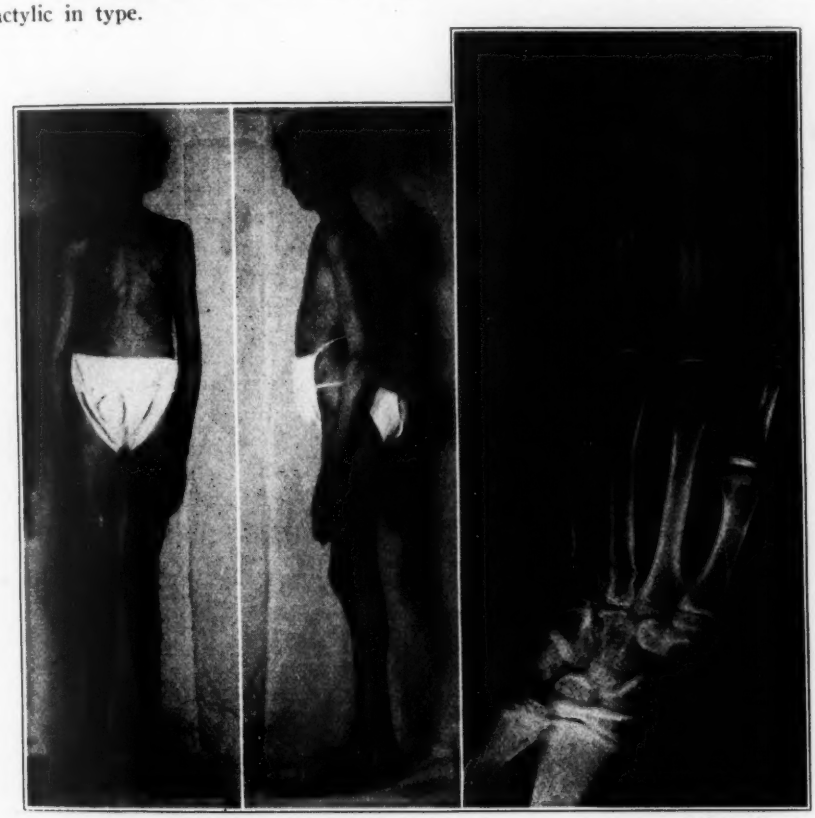

Fig. 10.—Marie C., aged 12, is a sister of Clarence C. (case 5). She is 5 feet, 4 inches tall (162.5 cm.) and weighs 85 pounds (38.6 Kg.). There are the characteristic spinal deformity and absence of adipose tissue. The roentgenogram of the left hand shows the characteristic length.

Vision in the right eye was 20/100 and in the left 1/100. The eyes were deeply set. The left eye diverged 20 degrees. The pupils were slightly eccentric to the temporal side and obliquely oval, each measuring 3 by 4 mm. The irides were very tremulous and dilated fairly well with atropine. The stroma of the iris was apparently normal. The lenses were partially and uniformly opaque, the right lens being dislocated upward and temporalward; the left, upward and nasalward. While the patient was in the hospital each lens was needled several times, it being necessary to transfix each lens and perform a posterior capsulotomy. There was

ultimately successful absorption, and on discharge the vision was as follows: In the right eye, with +6.50 sph. ⊃ +4.50 cyl., axis 120°, it was 20/40. In the left eye, with +6.75 sph. ⊃ +3.00 cyl., axis 60°, it was 20/40. With a +3.00 sph., right and left, Jaeger's test type 0.75 was read.

General treatment with sunbaths, forced feeding, cod liver oil and fruit juices over a period of three months resulted in a considerable improvement in the nutrition and a slight improvement in the amyotonia. On March 6, 1935, the height was 57½ inches (146 cm.) and the weight 67 pounds (30.4 Kg.). The boy had attained the third grade in school. Examination by Dr. A. R. Hall revealed no cardiac murmurs or any other abnormalities.

Summary.—This patient presented the mildest type of arachnodactyly, evidenced principally in the amyotonia and general muscular underdevelopment. There existed a slight similar tendency in the father and definite arachnodactyly in one sister. Four of the six members of the family showed ectopia lentis.

Case 6.—Robert S., aged 15, referred by Dr. Frank Brabec, was admitted to the Miller Hospital, St. Paul, on March 21, 1934, with visual impairment, cardiac symptoms and asthenia. He was the son of the patient to be reported on next (case 7).

His paternal grandmother had long bones and died of a cardiac disorder. One brother was living and well. One brother died suddenly of cardiac disease at the age of 16. One sister died of pneumonia. Another sister was typically arachnodactylic, with ectopia lentis (case 8), and a third sister was living and well. Four of the six children could not be classified as arachnodactylic; the paternal grandmother, father, one sister and this boy were all arachnodactylic. Defective vision had been present since infancy. The right eye became totally blind as a result of retinal detachment at the age of 10. The boy had reached the eighth grade. He read a great deal, because the cardiac disorder precluded any strenuous activity. Only during the past year had he been dyspneic, with palpitation, general fatigue and cyanosis of the fingers after slight exertion. Rickets had developed when he was an infant. His intelligence was excellent.

Examination by Dr. A. R. Hall, on March 24, 1934, showed that the boy's appearance was hydrocephalic, with marked frontal bosses, a broad forehead, a long face and a narrow, retracted chin. The hair was normal. He was a mouth-breather, with hypertrophied tonsils, irregular teeth and a high alveolar arch. No glandular disorder was apparent. The auricles appeared normal. The chest was long and flat, with precordial bulging and visible pulsation in the fifth interspace outside the nipple line. The abdomen was flat, and there was no pubic hair. The genitalia were normal. The reflexes were normal.

The boy's posture was faulty, and he was not tall, probably because of the spinal curvature. He weighed 90 pounds (40.8 Kg.). He stood with a slight bending to the right, the arms extending to within 2 inches (6 cm.) of the knees. The lower margin of the thorax approximated the crest of the ilium. The lumbar portion of the spine showed marked rotation with scoliosis to the left, the concavity being directed to the right.

The upper extremities, besides being abnormally long, had a permanent flexion of the elbows of about 5 degrees. The hands and fingers were long and thin. The musculature of the lower extremities was poor, and the muscle tone was very poor. The knee and ankle joints were relaxed, the knees being hyperextended and deformed. The feet were downflexed, with bilateral valgus. The feet and toes were abnormally long, the great toes measuring 2¾ inches (7 cm.). The boy wore size 8 shoes.

Roentgen examination showed lateral spinal curvature with the maximum convexity to the right in the midlumbar portion of the spine and with rotation of the bodies of the vertebrae, resulting in asymmetry of the pelvis.

The heart was shown, by a roentgenogram taken at a distance of 6 feet (183 cm.), to extend 9.3 cm. to the left and 3.3 cm. to the right of the median line, the total transverse diameter of the heart being 47 per cent of the total inside diameter of the chest. There was definite elongation of the heart. At examination the heart rate was 76 and regular, and the apex beat was felt in the fifth interspace. There was no palpable thrill, but there was a distinct rather rough diastolic mur-

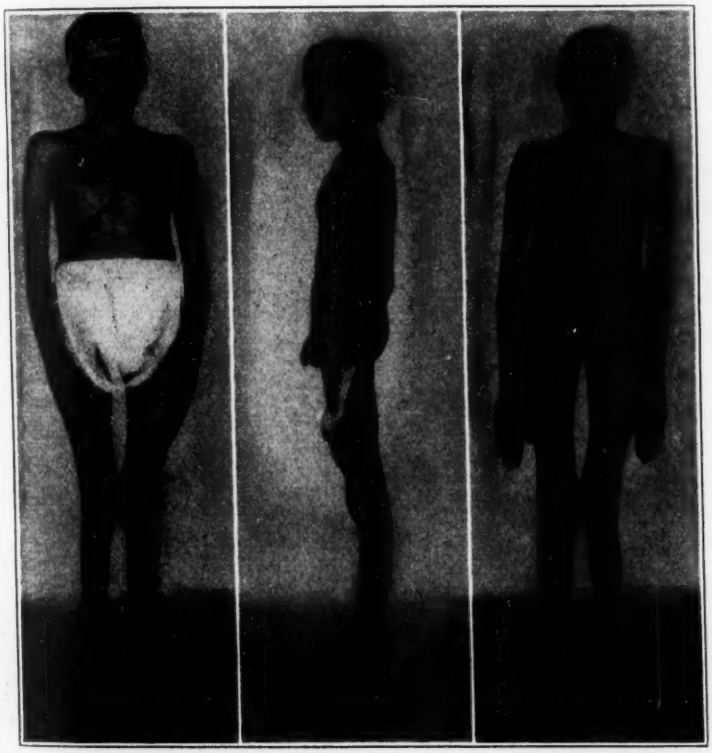

Fig. 11 (case 6).—R. S., aged 15, is blind in the right eye, owing to retinal detachment. Vision in the left eye with +10.00 sph. ⌒ +1.00 cyl., axis 70°, is 20/50. This patient shows spinal deformity, muscular atrophy, an absence of adipose tissue, elongated extremities and serious cardiac impairment.

mur over the entire cardiac area, best heard in the pulmonary area. No pistol shot sound was heard over the brachial or the femoral artery. The blood pressure was 80 systolic and 55 diastolic.

The rather rough tone, the absence of any increase in the pulse pressure and the absence of a pistol shot sound over the arteries were against a diagnosis of aortic regurgitation. It is possible, however, that there was a congenital defect at the orifice of the pulmonary artery. There was no clubbing of the fingers. Urinalysis showed a trace of albumin but no casts. Serologic examination revealed no abnormality.

The eyes were not particularly sunken, and there was not the extreme absence of fat that has been observed in other patients. The right eye diverged 20 degrees.

Vision in the left eye was 6/150 with the minus lens; without the lens the patient could read Jaeger's test type 1.75 at 4 inches (10 cm.). The right pupil was dilated to 6 mm. The diameter of the left pupil was 4 mm. in normal light and 8 mm. under the influence of homatropine. The irides were tremulous; the stroma appeared normal; and the lens of each eye was ectopic downward and temporalward but not opaque. With undilated pupil the vision in the left eye, with +10.00 sph. ⌒ + 1.00 cyl., axis 70°, was 20/50. Near vision was not improved

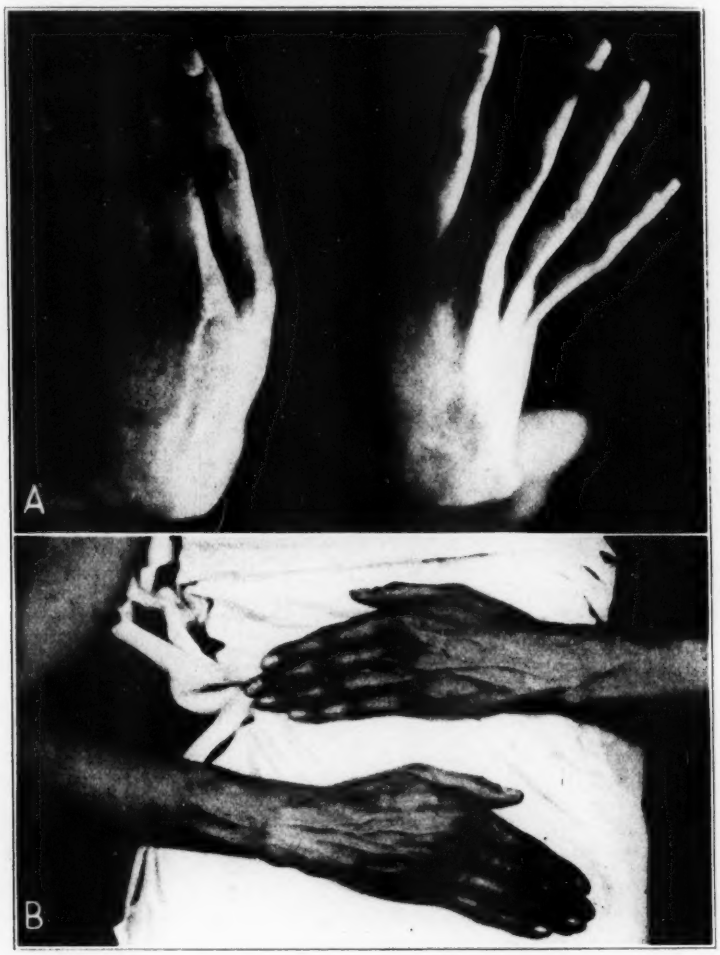

Fig. 12.—*A*, the arachnodactylic hands of H. S. (case 7), father of R. S. (case 6, fig. 6). He is 6 feet, 3¼ inches (191 cm.) tall and wears size 11 shoes. The heart is impaired. *B*, arachnodactylic hands of M. S. (case 8), the 21 year old sister of R. S. (case 6) and the daughter of H. S (case 7, fig. 12 *A*). She has extremely long fingers and toes.

by an added spherical lens when the pupil was contracted, but when the pupil was dilated, the patient preferred +3.00 sph. added and read Jaeger's test type 1.25. Ordinarily he read better without glasses. The left optic nerve and retina appeared normal. .

Summary.—This patient exhibited many of the common characteristics of arachnodactyly, with marked prominence of the ocular and cardiac symptoms. There was a suggestion that the condition was hereditary.

CASE 7.—Hjalmar T. S., aged 50, the father of Robert S. (case 6) and of Muriel S. (case 8), was referred by Dr. Frank Brabec on Feb. 25, 1935, on account of defective vision. His mother and father, five brothers and a sister were all over 6 feet in height but had fair vision; none had any cardiac disorder. His own son and daughter had eyes similar to his own and had cardiac disease.

Physical examination by Dr. A. R. Hall showed the patient's height to be 6 feet, 3¼ inches (191 cm.) and the weight 154 pounds (69.9 Kg.). At one time the patient's weight had reached 180 pounds (81.6 Kg.). Aside from the defective vision, his complaints related to frequent headaches and frequent dyspnea. His appearance was not strikingly abnormal, but the long, flat-chested body with scoliosis to the right in the lower dorsal vertebrae, with some torsion of the vertebrae and with a definite depression in the lower part of the sternum, was extremely suggestive. The length of the arms and legs was not disproportionate to the body length, but the hands and feet, especially the fingers and toes, were typical of Marfan's syndrome. He wore size 11 shoes.

The head was dolichocephalic, with narrow mandibles and a highly arched palate. The thyroid gland and the lungs were normal. The heart was dislocated to the left by the moderate *Trichterbrust*, the right cardiac border lying at about the level of the mesosternum and the left border to the left of the midclavicular line. The total diameter of the heart was about 12 cm., the apex being in the fifth interspace. The pulse rate was 78. The blood pressure was 140 systolic and 55 diastolic. No thrills were in evidence. The action of the heart was regular, with a soft, short systolic murmur at the apex that was not transmitted far. There was a soft systolic murmur at the base and also a marked diastolic murmur in the aortic region, diagnostic of aortic regurgitation. The reflexes and coordination were normal. The abdomen was normal except that the costal borders approximated the iliac crests.

The patient had had poor vision since infancy and had worn glasses since he was 8 years of age. Vision in the right eye had been lost suddenly when he was 30. Examination revealed that the right eye diverged 30 degrees and that there was a complete retinal detachment. In the left eye he wore —3.5 sph. ⊂ —2.75 cyl., axis 45°, and with it obtained vision of 17/200. He was able to read Jaeger's test type 1.50 at 4 inches (10 cm.). There was no record of the condition of the iris or pupil except that the pupil reacted to light normally and both pupils dilated incompletely.

Both lenses were dislocated upward and showed definite opacities. The left fundus showed no marked abnormalities and no myopic crescents.

Summary.—This patient did not show the marked changes that were present in his daughter and son, but the picture was sufficiently characteristic to be classified as arachnodactyly with ectopia lentis.

CASE 8.—Muriel S., aged 21, daughter of Hjalmar T. S. (case 7) and sister of Robert S. (case 6), was referred by Dr. Brabec on Feb. 25, 1935, on account of her eyes. The family history is given in the report of case 6. There was no special physical complaint except that she was reported to have some cardiac disorder.

Birth had been normal at full term. She was a fat, healthy baby and began to walk when about 1 year old. She started to attend school at 8 years of age and went as far as the eighth grade with better than average marks. Apparently

all the changes in growth began during early childhood, although at no definite period were they remarked on, as all the members of her family were large. Apparently the cardiac symptoms were not prominent until she had chorea at 17. She had previously had measles and influenza.

Physical examination by Dr. A. R. Hall revealed that the height was 5 feet, 9¾ inches (177 cm.) and the weight 111 pounds (50 Kg.). She was an exceedingly tall, slight girl with excessively long toes and fingers. The entire musculature was poorly developed, and there was a decided absence of subcutaneous fat. The history reveals that she had been physically weak since adolescence. A postural defect was present, due to kyphosis of the spine in the upper portion of the lumbar region. The appearance of the face and skull was typical. The head was dolichocephalic, with narrow jaws and a very high palate. The skin was normal. Apparently the thyroid gland was normal, but a basal metabolism test was not given. The menstrual history was normal. The chest was long and flat, with moderate *Trichterbrust*. The lungs were normal. The pulse rate was 104. A presystolic murmur at the apex, a marked diastolic murmur in the aortic area and an accentuated second sound in the pulmonary area established a diagnosis of organic mitral stenosis and aortic regurgitation. Apparently there was no cardiac hypertrophy.

The appearance of the limbs was characteristic, especially that of the hands and feet. The second finger of each hand measured 12.5 cm. from the metacarpophalangeal joint to the tip of the finger.

The mentality was excellent. The neurologic examination gave essentially normal results. The reflexes and coordination were normal. Laboratory tests showed no abnormalities.

Vision in the right eye was 7/200; with —12.00 sph., 10/200. In the left eye it was 20/200; with —12.00 sph., 20/200. She was able to read Jaeger's test type 0.50 at 3 inches with each eye, either with or without glasses. Iridodonesis was present. The pupils were only moderately contracted and dilated well under the influence of atropine. The iris pattern was normal. Both lenses were dislocated upward and nasalward fairly equally. The vitreous showed degenerative changes, and, notwithstanding the high myopia, there were no crescents. Any modification of the correction which she had worn since the age of 8 did not improve the vision. The dislocation upward and nasalward was exactly the same as that seen in the father but unlike that of the brother, in which the ectopia was downward and temporalward.

Summary.—This girl presented the ordinary picture of arachnodactyly with ectopia, dolichocephaly, a spinal defect, muscular dystrophy and a cardiac lesion, the origin of which was uncertain, in view of the history of chorea.

COMMENT ON THE ETIOLOGY

The interesting feature in the association of ectopia lentis and arachnodactyly is the problem of the etiology. The phenomenon of arachnodactyly is undoubtedly due to a mesoblastic growth anomaly; at least, only mesoblastic tissues, with the exception of the eyes, are involved. The suspensory ligament of the lens is probably of ectodermal origin.

Many theories of causation have been suggested for arachnodactyly. Among these are the theories that the condition is due (1) to an

endocrine disturbance, (2) to a form of growth disturbance allied to mongolism, (3) to muscular dystrophy and (4) to embryologic nutritional disturbances.

Little is known of the endocrinology of the embryo, whether endocrine secretional ability is an independent function of the fetus or whether the secretions are supplied through the mother. Assuming that arachnodactyly is of endocrine origin, it would seem possible that owing to some cause effective during intra-uterine life the fetus receives an abnormally increased amount of pituitary secretion, either from its own gland or from that of the mother. The condition develops slowly, and sometimes the changes are not observed until childhood, when the influence of the pituitary gland is more dominant. Weill, in reviewing all the evidences of the endocrine origin of arachnodactyly, said:

> From the clinical point of view, taking into consideration our actual knowledge of the repercussions provoked by endocrine gland deficiency, it would seem very probable that there is a relationship of cause and effect between modifications of these glands and the multiple malformations which we have mentioned. All these causes of dystrophies can demonstrate their influence upon the development of the embryo in general, and on that of the endocrine glands in particular, without it being possible in the actual state of our present knowledge to discern either their nature or the part that each plays.

The theory that arachnodactyly is a form of mongolism has slight support. In arachnodactyly the mentality is usually not affected. The head is usually dolichocephalic, while that of the mongol is brachycephalic and devoid of cranial eminences; the mongolian hand has thick rather than thin fingers. The mongolian idiot is usually regarded as a product of exhaustion, coming as the last of a very large family.

Thursfield, in 1917, according to Young,

> was the first to lay stress upon the atony and poor development of the entire musculature as a prime etiologic factor in arachnodactyly, though he admits that undue length and slenderness of the extremities do not occur with muscular atony. . . . Amyotonia is not usually familial; though its onset is generally intrauterine, and it is accompanied by many of the abnormalities noted in archanodactyly.

Young stated that "bilateral dislocation of the lens is not recorded in cases of amyotonia by Reuben and other writers." Moreover, the Charcot-Marie-Tooth phenomena are not usually observed in arachnodactyly. One cannot ignore the fact that the muscles are affected in about 80 per cent of cases of arachnodactyly; that the amyotonic state is commonly observed with arachnodactyly but is nonprogressive, which is not true in most of the cases of congenital dystrophies. One must consider the influence of endocrine or metabolic dysfunction in both these conditions and the possibility that the same underlying cause or causes prevail.

One still must explain particularly the changes affecting the suspensory ligament of the lens, which is supposedly of ectodermal origin. According to Weve, the ocular malformations are secondary and are provoked by primary lesions of the mesoderm.

In a recent personal communication, Dr. Ida Mann, a recognized authority on embryology who has given some thought to this subject, in discussing possible causes of colobomas of the iris, choroid, ciliary body and suspensory ligament, stated:

> These anomalies may all arise in at least two ways: first, by primary aberrant growth of ectoderm. This refers to those cases which show evidence of abnormal eversion of the inner layer of the optic cup along the foetal fissure, and the defects are in the lower part of the eye. Second, they may arise by undue persistence of mesoderm, which refers to cases showing abnormal persistence of some part of the hyaloid blood system and the defects may be in any sector of the cup including the "typical" position below.

In considering the possible causes of ectopia lentis, Dr. Mann stated:

> These may be:
>
> 1. Failure of formation of the zonule fibres at one part owing to persistence of capsulo-pupillary vessels. This is more likely to produce coloboma than ectopia.
>
> 2. Undue length of zonule fibres on one side with shortening on the other but no gap. This would be a primary ectodermal anomaly.
>
> 3. Simple decentration of the lens and optic cup margin on each other. The reason for this is not known, but it might be considered as analogous to the decentration of the pupil and cornea found in corectopia; all the parts may be normal but their relative positions are wrong.

Further, in considering the factors involved whereby several congenital defects are linked in a definite syndrome, she wrote to me:

> One has to remember that various factors may account for the linking of defects. In the first place, the defect may be in a certain chromosome. Since the chromosome carries many genes mediating many different characters in various parts and various tissues, it is to be expected that superficially unrelated characters may be found to be related by their genic position in the blastomeres. There is no evidence to lead us to suppose that mesodermal and ectodermal structures are segregated in different chromosomes, and there is no inherent reason why the characters for the extremities should not be carried by the same chromosomes as certain eye characters. Indeed there is even slight evidence in favour of their being in proximity, since poly- and syn-dactyly are common concomitants of many ocular anomalies (macular coloboma, anophthalmos, oxycephaly and others) . . .
>
> In the second place, if the defect is not genic it is possibly due to some toxic or endocrine disturbance acting at a later date. There is nothing inherently improbable in such a condition affecting both mesodermal and ectodermal structures alike, since the action of toxins during development is specific, not for the toxin, nor for a given tissue, but for the period of development at which it begins to act and for those structures which are undergoing most rapid differentiation at the time. Thus lamellar cataract is associated with changes in the teeth (ecto-

derm) and in the bones (mesoderm). A depressant acting soon after the third month might conceivably act on the outgrowth of zonule fibers and on parts of the skeleton as well, if both were actively growing at the time.

If one considers the matter of ectopia lentis alone, the etiology could easily be explained on the basis of persistence of the nutritional blood vessels of the embryonic lens. When one comes to consider the association of ectopia lentis with arachnodactyly it is difficult to escape the thought, in view of the familial and hereditary trends so frequently found, that the genic possibilities deserve serious consideration. Even though one may theorize indefinitely regarding the etiology of this well established association from a clinical point of view, one must conclude that any theory regarding ectopia lentis should include also a general consideration of the patient and not alone an ophthalmologic study.

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ABSTRACT OF DISCUSSION

DR. WILLIAM ZENTMAYER, Philadelphia: The report of Sorsby in 1935 entitled "Congenital Bilateral Coloboma of the Macula Associated with Apical Dystrophy of the Hands and Feet," with an account of the familial occurrence of the condition, is of interest in connection with the theory that the Marfan syndrome is due to a fault in the mesodermal anlage, as in the Sorsby syndrome only mesodermal structures are involved. To me a mesodermal fault seems to be a reasonable explanation of arachnodactyly. Only the fact that the dilator muscle of the iris and the zonule of the lens, which are at times involved, are not mesodermal in origin has stood in the way of its general acceptance.

It seems fair to assume that the extreme stretching of the coats of the eyeball, which must be present in eyes with myopia as high as 60 diopters, may cause a rupture of the zonular fibers and thus in some instances result in ectopia lentis. One may find a further explanation for the displacement of the lens in the assumption made by Collins and Hess that it may be occasioned when the adhesions between the ciliary body and the margin of the lens are denser and less elastic on one side than on the other. Collins further stated that the arrest of development of the iris, ciliary body and suspensory ligament in a portion of their circumference is probably due to some abnormal adhesion, thickening or prolonged persistence of the lateral prolongation forward of the fibrovascular sheath of the lens.

So far as I know, there has been no histologic study of the eye in a case of arachnodactyly. That there may be a congenital absence of the dilator muscle has been demonstrated in cases of congenital miosis by Holth and Berner.

It is assumed that the miosis and the rigidity of the pupil to mydriatics in cases of arachnodactyly are due to hypogenesis of the dilator muscle. Lloyd has suggested that the unyielding pupil is caused by the rigid framework of the iris, and Vail has stated that in cases of arachnodactyly with megalocornea there is atrophy of the superficial layers of the iris which is continuous, leading from the border of the pupil and extending to the angle. It is possible that irritation of the dislocated lens also may contribute to the smallness of the pupil.

According to Macht, the action of atropine in dilating the pupil is due almost entirely to paralysis of the sphincter, whereas that of homatropine is not entirely due to paralysis of the parasympathetic action but is probably at least in part to be explained by the direct action on the muscle cells themselves. The work of Macht, however, requires confirmation. If it is true, this difference in the action of the two mydriatics might assist in determining whether the dilator muscle is absent, as one might expect decidedly less mydriasis from homatropine than from atropine if the dilator muscle is absent.

DR. RALPH I. LLOYD, Brooklyn: Arachnodactyly is of interest because the eyes show dislocation of the lenses or miosis or both. Dislocations of the lens are of three types: The most frequent type occurs secondary to intra-ocular disease; the second type is of traumatic origin, and the third has been considered an entity without associated changes elsewhere but recognized as familial and inheritable at times. The slit lamp has shown that the zonular fibers in the third type of case are loose, elongated, ruptured or even absent in part of the circumference of the lens. As a result of lowered or locally deficient zonular tension the lens assumes a convex form equivalent to extreme accommodation, explaining the high myopia regularly found in these cases: 14 diopters in one of mine. Through the aphakic pupil the refraction in the case reported is from plus 2 to 7 diopters, indicating that the eyeball is short. The extreme curve of the anterior surface of the lens can be appreciated with a binocular loupe and has been designated spherophakia. In a few instances the lens has been removed from the anterior chamber and has shown a coloboma at one or more places, which is ascribed to the lack of zonular tension during the growth of the lens. Thus far all features can be attributed to an imperfect development of the fibers of the zonula which do not arise from the mesoderm.

The slit lamp shows also pigment deposits on the zonular fibers, opacities of the vitreous and tapetoretinal degeneration, even in young patients. In addition to these changes in the lens and uvea, a cornea with a transverse diameter larger than 12.5 mm. has been found and in some cases the other symptoms of hydrophthalmos also. I have found one case of microcornea but none of megalocornea. It is unusual to find a marked malformation of the eye unassociated with other defects. Is it not reasonable to think that a lesser degree of zonular disease would cause weakness of the fibers only, which in turn would allow the lens to assume habitually a more nearly spherical form, causing the myopia, while the uveal changes and the changes in the tapetum would explain the late progressive changes of high myopia, such as elongation of the posterior half of the eyeball and chorioretinal degeneration, so regularly found in older patients with high myopia? All these conditions are hereditary and familial.

Like arachnodactyly, status dysraphicus was first described by neurologists and pediatricians as an entity some time before its ocular features were recognized. Passow called attention to the fact that syringomyelia is a familial condition and that lesser forms of the disease are much more common than the typical, better known and fully developed condition. Traced back to the early days of the embryo, there is defective formation of the neural tube. The primitive central spinal canal is surrounded by cells that have a great deal to do with the trophic

control of the body. In syringomyelia these cells suffer early and severe damage. In status dysraphicus clinicians look for some of the milder signs that are found in typical syringomyelia. Among these is a peculiar crooking of the fingers, which may persist only in the little fingers. In women the breast on the affected side is much smaller than that on the opposite side. Spinal curvatures, weak muscles and long thin bones are present as in arachnodactyly. With the hands raised horizontally at the sides the distance from the tip of the longest finger of one hand to that of the other hand should be exactly the same as the person's height. In status dysraphicus this span is often several centimeters more than the height. In addition, the arms and hands are sometimes disproportionately large as well as long.

Another developmental error is a sternal groove, which is sometimes so pronounced that the heart may be displaced to the left. In addition to dislocation of the lenses, Horner's syndrome and heterochromia may be found on the same side as the smaller breast. These features lead one to take a broader view of congenital familial luxation of the lenses and associated skeletal changes; for, reasonable as Weve's explanation of the condition as a mesodermal dystrophy seems to be, Vogt's argument that it is a coupling of developmental errors in which the chromosomes are concerned is much more reasonable. The older idea of heredity explained tuberculosis and cancer as a direct transmission of the virus or a lowered vitality which invited the invasion of an infecting agent. While it must be admitted that Spirochaeta pallida may pass from mother to child, modern studies of heredity have shown that many diseases that were previously considered separate entities are really familial and hereditary degenerations and obey the mendelian law. A study of the chromosomes will probably answer at least some of the questions.

Dr. M. N. Beigelman, Los Angeles: I wish to report on a group of twelve patients with congenital ectopia of the lens and arachnodactyly who have been observed for a period of nine years. This group consists of two interrelated families. The first family originated through an intermarriage of second cousins, both having arachnodactyly and ectopia of the lens. Three of the four sons have the same condition. Only one child in the third generation is free from the syndrome. The grandmother of this family has a sister suffering from combined arachnodactyly and ectopia of the lens. Her daughter and one of the two grandchildren have the same condition.

The characteristic physical features in this group of cases are lack of subcutaneous fat, inadequate development of the musculature, curvature of the spine and enlargement of the hands and feet. The appearances of all these persons are strikingly identical. Incidentally, an endocrinologic study of this family, performed by Dr. S. Glass, did not reveal any glandular abnormality.

The grandparents, 72 and 61 years old, respectively, both have complete dislocation of the lenses into the vitreous. Two of the four brothers in the second generation have lenses lost in the vitreous, while in the third generation only partial ectopia of the lens is reported.

This brings out clearly the point that partial ectopia of the lens has a tendency to become complete in time, and with it various complica-

tions affecting the visual functions are likely to occur. In other words, while ectopia of the lens is a congenital anomaly, it is at the same time a clinically progressive disease. This fact, combined with strong hereditary tendencies, raises the question as to whether one should remain indifferent to a continuation and perpetuation of this defect or whether some sort of educational work is indicated in the afflicted families.

Dr. Edward Jackson, Denver: Since Marfan's syndrome has been recognized and accurately described, I have seen no instance of it. But I recall two patients with ectopia lentis who were under observation for several years and whose status seemed to indicate the practical importance of the general recognition of this condition. These were cases in which, like those presented by Dr. Beigelman, there was progressive dislocation of the lens. The patients were not relatives. They were watched from the ages of 8 and 11 years to 16 and 20 years, respectively. The dislocation of the lens was such that at first it could be easily overlooked. But the fact that the edge of the lens was behind the small pupil gave considerable myopia, for which advice was sought. The general features of tallness and slenderness and the extreme length of the hands and feet were observed, and I cannot help thinking that these patients belonged to the class under discussion. Since, as the last speaker has pointed out, dislocations of the lens in general tend to become progressively worse and more and more complete, early recognition of the syndrome is of practical importance.

AN ADVANCED STAGE OF DIKTYOMA

REPORT OF A CASE

PETER S. SOUDAKOFF, M.D.
PEIPING, CHINA

In 1908 Fuchs,[1] in his paper entitled "Proliferations and Tumors of the Epithelium of the Ciliary Body," described four instances collected from the literature of a tumor which he called diktyoma. Since that time several additional cases have been reported by various authors, but still the total number does not exceed eight.

This case is reported because of the great rarity of the disease and the advanced stage of involvement of the eyeball.

REPORT OF CASE

W. Y. H., a Chinese farmer aged 28, came to the St. Michel Hospital in Peiping on Dec. 11, 1934, complaining of loss of vision of the left eye. The patient was referred to the ophthalmologic clinic of the Peiping Union Medical College for consultation. There he gave the following history: Ten months prior to consultation the vision of the left eye began to fail, and in the course of two months it was completely lost. Six months later the eyeball began to protrude, a process which was associated with pain in the eye and headache on the left side.

On examination his right eye was found to be normal. The vision was 6/6, and he could read Jaeger's test type no. 1. The most prominent part of the left eye projected 15 mm. beyond the plane of the right cornea. The eyeball was also turned upward so that the cornea was entirely covered by the upper lid. The cornea was clear; the anterior chamber was very shallow, and the iris showed partial ectropion uveae. The pupil was widely dilated and fixed. The lens was cataractous. There was moderate lagophthalmos, and the part of the sclera corresponding to the palpebral fissure was ulcerated. Tension was plus three. Vision was absent. No red reflex could be obtained from the fundus. The eyeball was freely movable. The preauricular glands were not enlarged.

I performed enucleation of the left eyeball on December 21 at the St. Michel Hospital, with the assistance of Dr. Bussière, who referred the case to me and gave permission to use the eyeball for study. Two days before the operation, the ulcer of the sclera below the cornea perforated, and bleeding occurred from the perforation. In spite of canthotomy a solid piece of the contents of the eyeball escaped through the perforation in the sclera during the delivery of the eyeball. The postoperative course was uneventful. The patient presented himself on Jan.

From the Department of Ophthalmology of the Peiping Union Medical College.

1. Fuchs, E.: Arch. f. Ophth. **68**:534, 1908.

31, 1936, in the ophthalmologic clinic of the Peiping Union Medical College. Clinical examination as well as roentgenograms of the left orbit revealed no sign of recurrence or metastasis.

The eyeball and the compact mass collected during the operation were fixed in Zenker's fluid and embedded in pyroxylin. The eyeball measured as follows: anteroposterior diameter, 30 mm.; vertical diameter, 32 mm., and horizontal diameter, 25 mm. The piece that had been expelled from the eyeball during enucleation was of firm consistency and measured 35 by 18 by 21 mm. Seven millimeters below the cornea perforation of the sclera, plugged by a blackish mass, was observed. Horizontal serial sections were made through the eyeball. Because of the escape of tissues from the eyeball during enucleation, the normal relation-

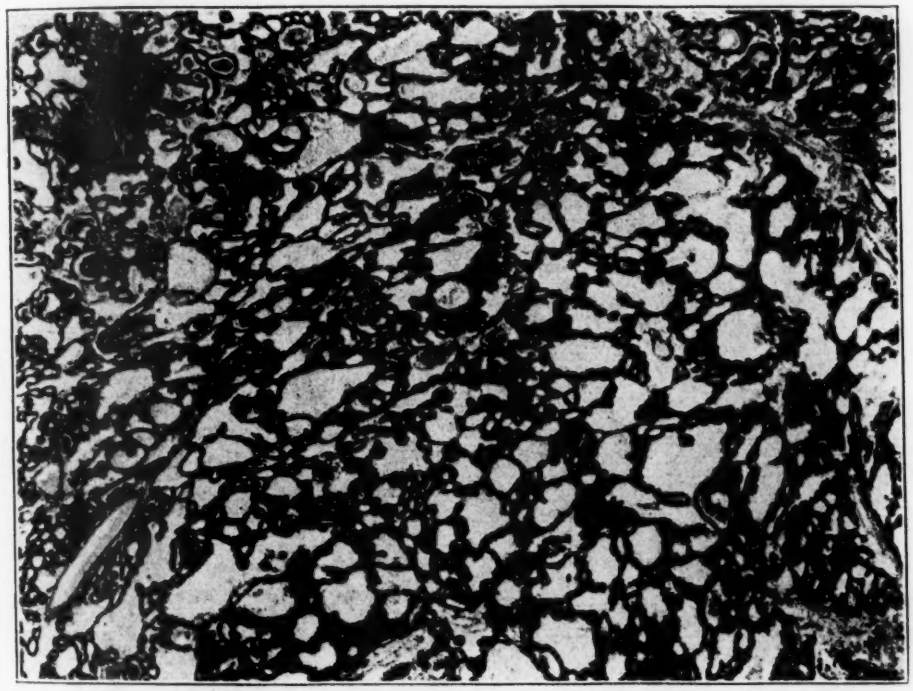

Fig. 1.—Photomicrograph (×24) showing the whole cavity of the eyeball filled with tumor cells growing chiefly in the form of bands, consisting of one or several layers of cells. In places tumor cells are arranged in groups.

ship between the structures was greatly altered. Some of the structures were observed in the delivered mass, i. e., the whole lens and part of the iris and ciliary body, while the other part of the iris and ciliary body as well as the choroid remained in the eyeball. Both the specimens contained tumors of similar structure. For the sake of convenience the two specimens will be described together.

Microscopic Examination.—The cornea was intact except for the lowest segment, which will be described later. Only a small portion of the ciliary body was observed in situ, while the greater part with the corresponding portion of the iris had been torn away and expelled from the eyeball. In the intra-ocular portion only a few ciliary processes and bundles of the ciliary muscle were preserved, while most of the stroma was transformed into edematous collagenous tissue. A

considerable part of the ciliary body was detached from the sclera by tumor cells growing between these two tissues. Expulsion of a part of the uvea during the operation apparently was facilitated by this growth. The portion of the iris which had been expelled from the eye was congested but free from tumor cells. The portion within the eyeball showed normal structure, except in its lower sector, where the posterior surface was invaded by tumor cells to a small extent. The lens contained cataractous globules in the subcapsular and cortical layers. The choroid was almost totally destroyed by tumor cells. The retina was represented by a few cells of the nuclear layers. A tumor occupied the whole cavity of the eyeball and had perforated the sclera in several places; below the cornea (as seen clinically), at the posterior pole (about 8 mm. in diameter) and at the temporal side.

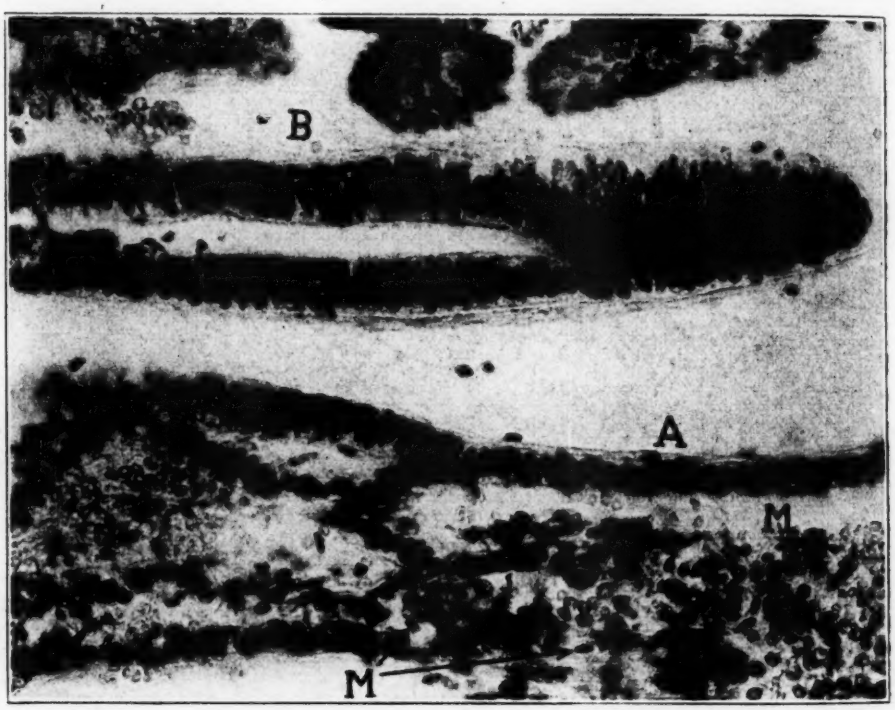

Fig. 2.—Photomicrograph (× 295) showing the tumor cells arranged in bands composed of one (*A*) or several (*B*) rows of cells. Mitotic figures (*M*) are present.

The tumor consisted chiefly of bands of cells, some of which contained several rows of nuclei and some, only one. The nuclei were oval and lay perpendicular to the long axis of the band. In these nuclei mitotic figures were common. The cell bands formed innumerable folds and convolutions and lined glandlike lumens, of varying size and shape. Some of the lumens were small and round, giving the impression of rosettes, while the larger looked like cysts. The lumens either were empty or contained coagulated fluid or a few cells resembling the tumor cells. Some contained capillaries (figs. 1 and 2).

The surface of the cell bands was formed by a rather distinct limiting membrane, next to which lay a protoplasmic rim free from nuclei. When the strands consisted of one layer of cells only, their borders were fairly distinct. In the multi-

cellular strands no cell outlines could be made out. In places the strands ran together and formed small solid accumulations of tumor cells. The individual tumor cell could be seen best where it invaded other tissues. It possessed an oval nucleus and two nucleoli with indistinct cytoplasm. On the whole, it looked much like the undifferentiated cells of the embryonic retina. The tumor possessed practically no supporting connective tissue. No glial tissue could be detected by special staining methods, nor were there any newly formed membranes or cartilaginous deposits.

The tumor mass contained vessels, mostly capillaries. Fresh and recent hemorrhages were scattered within the tumor, and there pyknotic cells were seen, but as a rule the tumor cells showed no tendency to necrosis, even if they were lying distant from the vessels. In general, the tumor cells were free from pigment. Pigment was noted only in the cells invading the structures which normally contain pigment or in the vicinity of these tissues. The tumor had broken through the sclera, destroyed a considerable part of it and grown behind the sclera. This part of the neoplasm was partly covered by a thin capsule made up of connective tissue, which contained pigment and numerous vessels.

The innermost lamellae of the lowest segment of the cornea were invaded by tumor cells, which were in continuity with those intruding on the adjacent part of the sclera. The sclera just below the cornea was heavily infiltrated by leukocytes and lymphocytes, and the scleral lamellae and nuclei were no more recognizable. Still farther below, the scleral tissue was entirely disintegrated, thus producing the large ulcer already described.

COMMENT

The case described is that of a tumor of the eyeball in a very advanced stage, the study of which was handicapped by the stage of the tumor and the disarrangement of the structure, owing to escape of a part of the eyeball during enucleation.

The tumor was composed of convoluted cell bands spreading in different planes and therefore appearing either as cylindric or oval tubes or as round formations (rosettes). In many sections it was fairly well seen that all these complicated convolutions were produced by the foldings and interlacings of a membrane (fig. 3). This observation is in full accord with Ginsberg's [2] description of the complexities of form which a diktyoma can assume. Each band is composed of one or several rows of cells with elongated nuclei lying perpendicular to the surface of the band. The solitary cells which invade the tissues possess a large oval nucleus and a small amount of protoplasm. In some places they accumulate in groups. The cells in the unicellular bands have the appearance of epithelial cells, while the cells lying free in the tissues and those making up multicellular bands resemble cells of the embryonic retina. Vacuolation of tumor cells, described by Fuchs, Martens, Merkel and Boeck, has not been observed in my case. Glial fibers have not been noted.

In consideration of the advanced stage of this tumor and the disorder of the tissues in the specimen, it is impossible to determine the

2. Ginsberg, S.: Uvea, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 1, p. 555.

original site of the growth. The type of the cells and their arrangement, however, were similar to those observed in a malignant growth derived from the nonpigmented epithelium of the ciliary body or pars ciliaris retinae, termed diktyoma. Malignancy in this case has been proved by the presence of numerous mitotic figures, usually observed on the inner side of the band, and by the destructive action of the growth on the ciliary body, choroid and sclera.

Fuchs [1] has divided the malignant tumors of the ciliary epithelium into two groups. The first group is characterized by the presence of cellular membranes composed of one or several layers of irregularly

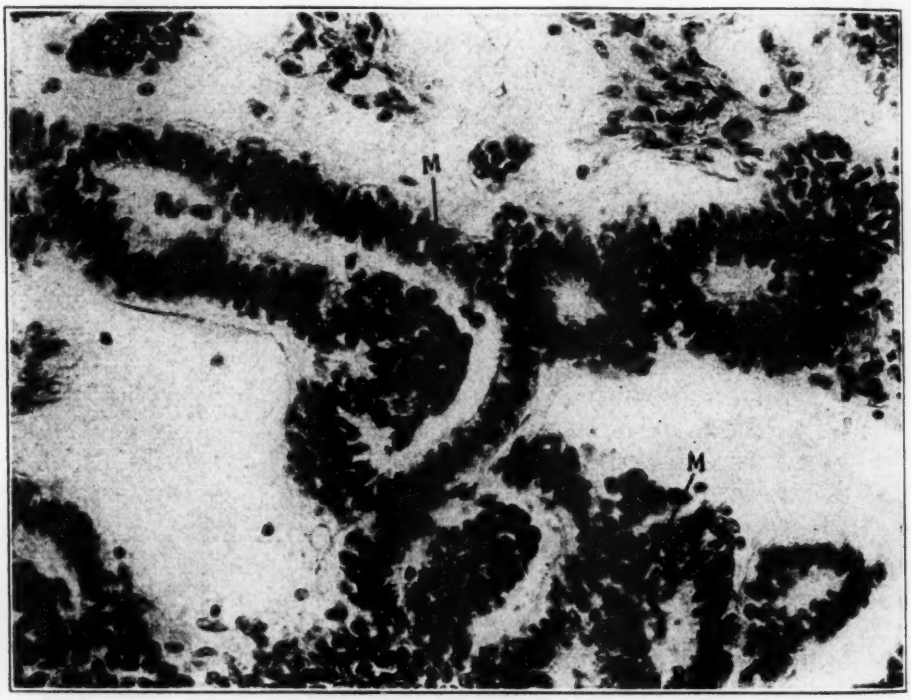

Fig. 3.—Photomicrograph (× 295) showing the folding and interlacing of the bands of the tumor cells to form tubules and rosettes. Mitotic figures (*M*) are common. Little connective tissue is seen between the bands.

arranged nuclei, resembling the embryonic retina before its differentiation into layers. For this reason, the tumor has been named diktyoma. This kind of tumor has been observed in children in the cases cited by Lagrange,[3] Leber and Emanuel,[4] Verhoeff,[5] Kuthe and Ginsberg,[6]

3. Lagrange, F.: Arch. d'opht. **12**:143, 1892.

4. Emanuel, C.: Virchows Arch. f. path. Anat. **161**:338, 1900.

5. Verhoeff, F.: Tr. Am. Ophth. Soc. **3**:351, 1904.

6. Kuthe, R., and Ginsberg, S.: Beitr. z. Augenh. Festschr. Julius Hirschberg, 1905, p. 127.

Greeves,[7] Velhagen [8] and Satanowsky,[9] with Boeck's [10] case of a 16 year old patient as the only exception.

The tumors of the second group, called malignant epithelioma of the ciliary body by Ginsberg, occur among adults. These tumors are usually pigmented and form either unicellular membranes or cellular tubes. They are often observed in patients who have previously had severe inflammation of the eye, resulting in the formation of connective tissue membranes. In this group belong the cases of Treacher Collins,[11] Fuchs,[1] Schlipp,[12] Meller,[13] Merkel,[14] Hine [15] and Martens.[16]

In the present case the tumor resembled embryonic retina so closely and answered the classic description given by Fuchs so accurately that the diagnosis diktyoma seems most appropriate, despite the age of the patient.

In all the other reported cases of diktyoma the eyeball was enucleated before extensive destruction took place, and one does not know what aspect the growth might have assumed if the eye had not been removed so early. Extensive destruction of the ocular structures has not been observed, but such a consequence of tumor activity is not impossible, as Fuchs pointed out. In China, where western medicine is gaining but slowly the confidence of the people, patients with diseases of the eye come for consultation with such advanced stages as are hardly ever, if at all, observed in Europe or the United States.

The growth in the present case thus represents the most advanced stage of diktyoma retinae so far reported.

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7. Greeves, A.: Tr. Ophth. Soc. U. Kingdom **31**:261, 1911.
8. Velhagen, C.: Klin. Monatsbl. f. Augenh. **58**:239, 1917.
9. Satanowsky, Paulina: Tumores epiteliales del cuerpo ciliar: Los diktyomas, Semana méd. **2**:1017 (Oct.) 1928; abstr., Zentralbl. f. Ophth. **21**:461, 1929.
10. Boeck, J.: Ztschr. f. Augenh. **69**:17, 1929.
11. Collins, E. Treacher: Tr. Ophth. Soc. U. Kingdom **11**:55, 1891; **14**:83, 1894.
12. Schlipp, R.: Arch. f. Ophth. **48**:353, 1899.
13. Meller, J.: Arch. f. Ophth. **85**:151, 1913.
14. Merkel, F.: Arch. f. Augenh. **97**:308, 1926; cited by Ginsberg.[2]
15. Hine, M.: Tr. Ophth. Soc. U. Kingdom **40**:146, 1920.
16. Martens, M.: Arch. f. Augenh. **89**:1, 1921.

TUMOR OF THE OPTIC NERVE

REPORT OF A CASE

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PORTLAND, ORE.

Primary tumor of the optic nerve is rare.[1] The growth may vary from a small localized thickening to a mass that fills the orbit and crowds the eye forward and literally out of the head. It is a condition essentially of early life and of slow growth, producing usually, as observed by Graefe, and exophthalmos straight forward, though sometimes with a deviation down and in.

The motility of the eye remains intact until a late period. The lids stretch as the eyeball advances and cover the cornea with a continually thinning structure. Later, when lagophthalmos and corneal ulceration have taken place, degeneration of the eyeball occurs rapidly. Hence, the earlier the growth is removed, the better the prognosis. Extension into the cerebral cavity sometimes occurs, but extension forward into the bulbus is unusual.

Ophthalmoscopically, one observes a neuritis of the postbulbar type, with venous engorgement. Later, evidences of atrophy of the optic nerve appear. The pressure of the growth from behind may produce flattening of the bulbus, marked shortening of the eyeball and consequent hyperopia. Retinal hemorrhages occur early, and blindness ensues.

The rate of development of the tumor, while variable, is ordinarily extremely slow. Weeks[2] quoted Finlay as saying that in fifty cases the period of development varied from two months to fifty years. Of twenty-five patients operated on, ten showed no recurrence and eight showed recurrence either locally or systemically. Postoperative deaths, most of them due to sepsis, numbered seven.

The growth originates either in the tissue of the nerve (intradural type) or in the capsule of the nerve (extradural type).

Parsons[3] cited reports of eighty-four cases of the intradural type of tumor collected by Byers, only four of which occurred after the twenty-fifth year of life. The swelling usually involves the posterior portion (two thirds or three fourths) of the nerve and varies from a slight localized thickening of the nerve to a pear-shaped mass filling the

1. Fuchs, E.: Textbook of Ophthalmology, ed. 5, Philadelphia, J. B. Lippincott Company, 1917, p. 631.

2. Weeks, J. E.: A Treatise on Diseases of the Eye, Philadelphia, Lea & Febiger, 1910.

3. Parsons, J. H.: The Pathology of the Eye, London, Hodder & Stoughton, 1904-1908.

entire orbit, sometimes even enveloping the globes. Section of the involved nerve shows a layer of proliferated cells beneath the dural sheath, i. e., between the dural and the pial sheath.

Formerly these tumors were thought to range through the gamut of endothelioma, neuroma, fibroma, sarcoma, myxoma, etc.[4] The same specimen often presents several phases of developing connective tissue. Parsons stated that all of them were of mesoblastic origin and probably congenital. He objected to the term "neuroma" for these tumors, as true neuromas "do not occur in the central nervous system of which the optic nerve is a part." So, too, he expressed the opinion that the term "glioma" is loosely used and is strictly applicable only to tissue which responds to the special neuroglial stains, rather than to all growths of connective tissue occurring in nerve structures.

While the intradural type of tumor of the optic nerve is rare, a tumor of extradural origin is even more uncommon. But reports of eighteen authentic cases (again quoting Parsons) have been published, only fourteen of which gave such details as age and sex. Of these fourteen cases, ten occurred in patients less than 21 years old and five in patients less than 10 years old. Ten were in males and six in females. The statistics regarding the sex of patients with intradural tumors showed that females predominated.

In general the extradural tumors are remarkably similar to the intradural variety, and Parsons expressed the belief that, except for the true gliomas, all arise from one or another of the nerve sheaths. Their anatomic peculiarities, slight malignancy and slow growth, he said, suggest a relationship to infective granuloma, while the possibility of local infection and also of parasitic influence should not be overlooked.

The problems involved in cases of this type are those connected with the diagnosis. Once the presence of a malignant condition is established or strongly suspected, prompt surgical treatment alone is indicated if one wishes to avoid a fatal outcome. Success depends on the size and the accessibility of the growth. In some instances it is possible to dissect off the tumor, leaving a functioning nerve and the eyeball in situ. More often it is necessary, as in the case to be detailed, to remove the bulbus with the tumor. A Krönlein incision may be required. If the neoplasm has extended to the periosteum, removal of the entire contents of the orbit is indicated.

In other words, surgical intervention aims first to preserve the bulbus, preferably with an incision through the soft tissues or if that is impracticable, with an incision through the bony tissues. But the growth in its entirety must be removed, even though it necessitates sacrifice of the eyeball and complete removal of the orbital contents.

4. Oberling, C., and Nordmann, J.: Les tumeurs du nerf optique, Ann. d'ocul. **164**:564 (Aug.) 1927.

The following is the report of a case of intradural tumor of the optic nerve.

REPORT OF CASE

V. S., an 11 year old girl, the seventh of eleven children (the mother was only 46), was sent in from the coast country on Jan. 2, 1934. She was a well developed child, though slight of build, of medium complexion and with a very delicate skin. No history of any eye trouble in the family or in the family of either parent was determinable. The mother stated that since the child was 5 the left eye had become more and more prominent and that the increase in size had been much more marked during the past four months. A reputable oculist was consulted when the condition was first noticed, but the diagnosis was left in doubt.

At the time of examination the left eye was markedly proptosed, the cornea being fully 2.5 cm. in advance of that of the right eye. The child could forcibly cover the eye with the lid but the mother said that during sleep the lids did not close. The cornea was still clear. The upper lid was greatly thinned. The right eye was normal in all particulars, including vision. The left eye retained no perception of light. There had never been any pain. There was no pulsation or bruit. Movement, though restricted by the exophthalmos (directly forward), was possible in all directions. There was no history of trauma and no enlargement of the thyroid gland. Tension by palpation seemed normal in each eye. A provisional diagnosis of tumor of the optic nerve was made. Roentgenograms and a general physical examination threw no light on the condition.

On Jan. 5, 1934, the consent of the parents having been obtained for the required surgical treatment, however radical, the child was operated on. After a wide external canthotomy it was possible to determine by palpation that the tumor filled the entire orbit, that it enveloped the nerve and that it was necessary to enucleate the eyeball. When the conjunctiva was dissected back the growth could be plainly seen. It was firm to touch and dark blue, and it filled the entire orbit, the anterior surface being even with the anterior orbital margin. From the center of the mass a section of the optic nerve protruded, connecting the neoplasm with the bulbus. This section of nerve was about ½ inch (1.3 cm.) long and appeared somewhat cystic on one side.

By slow and painstaking dissection the entire orbital contents were removed. As the orbital apex was approached a rather sharp hemorrhage arose, and to control it the mass was excised near the apex. The hemorrhage ceased, and the remaining portion of the tumor, a piece about the size of a robin's egg, was dissected out. The healing was uninterrupted, and recovery was prompt and without incident.

In view of the possibility of extension to the cerebrum it was deemed advisable to give the child postoperative roentgen treatments. She has now had three series of treatments at three month intervals. The orbit has healed perfectly, and the lids have taken on a normal appearance. The socket, however, is not adapted for a prosthesis.

Dr. Warren Hunter, assistant professor of pathology at the University of Oregon Medical School, has given much time and study to this case and prepared the skilfully mounted specimen showing half the tumor with the nerve traversing it.

On initial examination the tissue seemed to be that of a meningioma. The tumor was sent to the Army Medical Museum, where beautiful

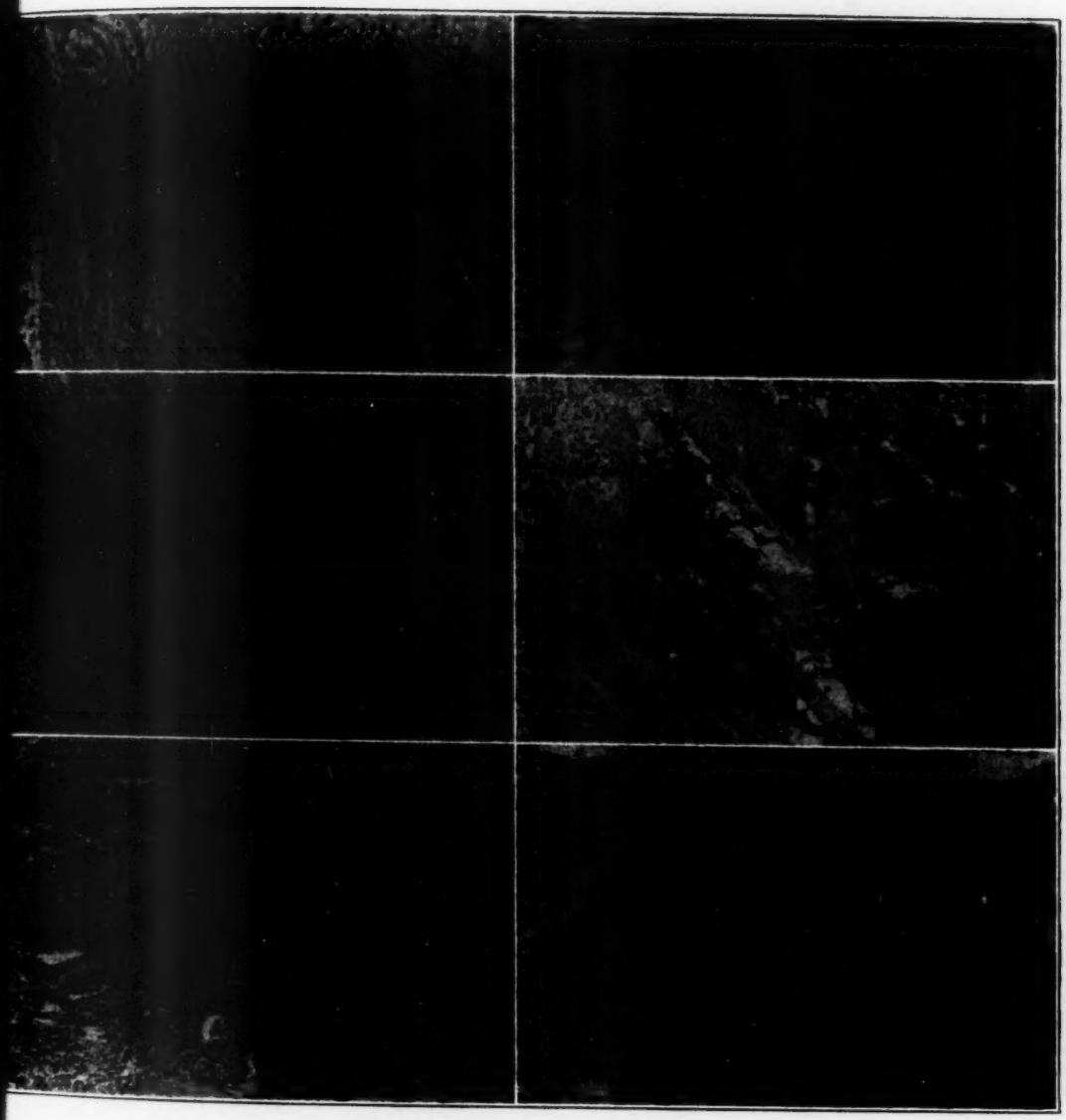

Fig. 1.—Glioma of the optic nerve.

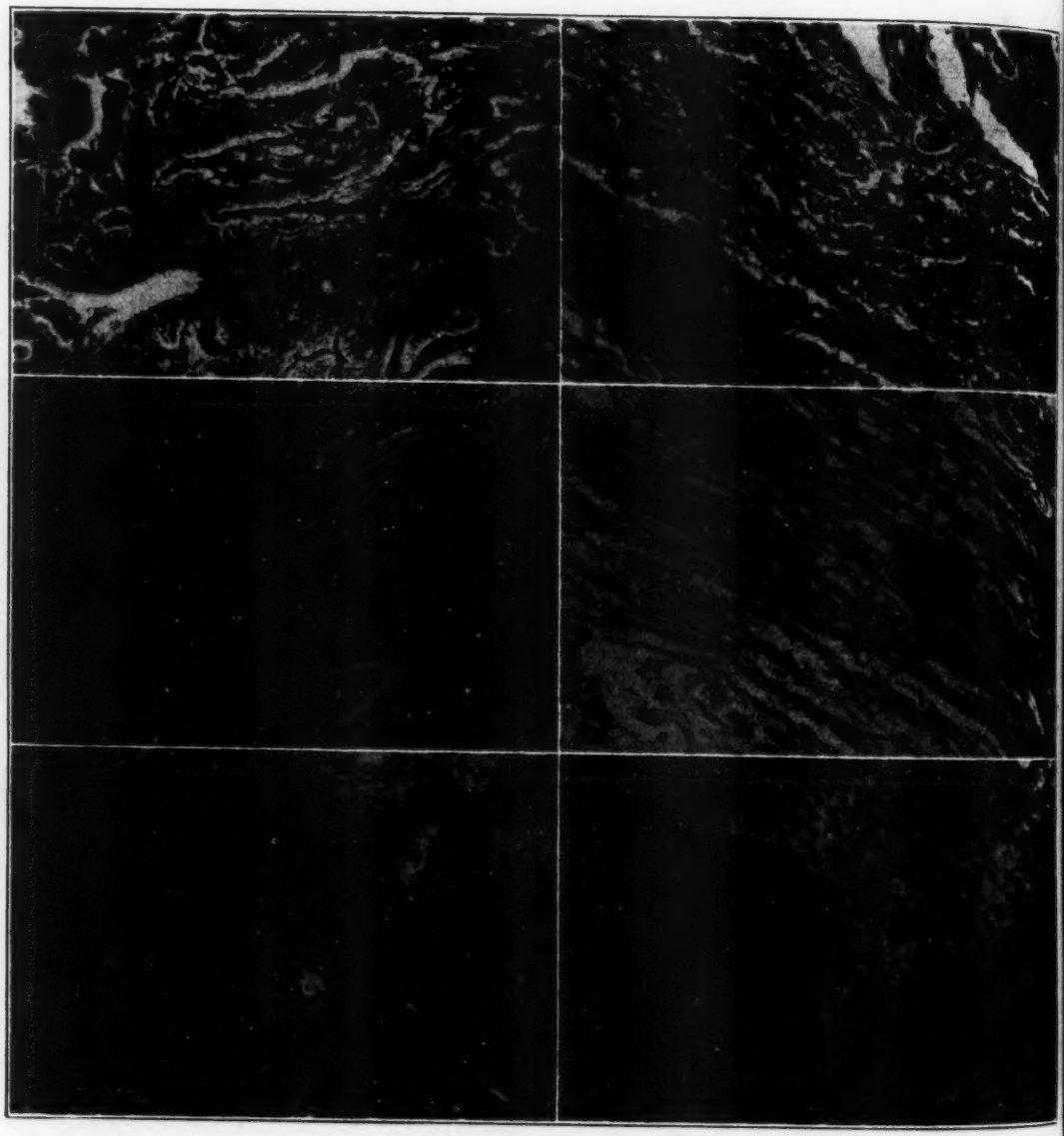

Fig. 2.—Glioma of the optic nerve.

sections were made by Mrs. Wilder and forwarded to Dr. Verhoeff, who rendered the following report:

The specimen shows a perfectly typical glioma of the optic nerve. It shows perhaps a somewhat unusual amount of interstitial hemorrhage, but hemorrhages are common in these tumors. The tumor contains many cytoid bodies which I have found to result from interstitial hemorrhages. I have recently described these tumors.[5]

In this diagnosis Dr. Hunter and Dr. A. J. McLean, clinical instructor in surgery and neuropathology, fully concurred, as shown in their report, which follows. They have also kindly furnished me with the accompanying photomicrographs.

The old preparations of the tumor (hematoxylin and eosin and Van Gieson stains) were reviewed. New preparations were made using the following stains: hematoxylin and eosin, Mallory's phosphotungstic acid hematoxylin, Van Gieson's and Hortega's silver lithium carbonate stain and Perdrau's stain for reticulin. The former description of the microscopic picture observed in sections prepared with hematoxylin and eosin and with Van Gieson's stain was confirmed. The swirling strands and general architecture shown in figure 1 *A* to *C* (hemotoxylin and eosin) are not uncommon in relatively acellular fibroblastic types of meningioma. The collagen shown by Van Gieson's stain was not confined to the nerve sheath and interstitial trabecula of the nerve (fig. 1*D*) but penetrated liberally and diffusely throughout the tumor, often without the slightest relation to the visible vessels (fig. 1*E*). Such mesodermal support (fig. 1*F*) is rare in glioma and not uncommon in many types of meningioma. That mesodermal support did not obtain throughout many highly cellular areas of the tumor was demonstrated, however, by the almost complete lack of reticulin in many parts, as shown by the Perdrau stain (fig. 2*A*), although it was abundant in the sheath (fig. 2*B*) and sharply demarcated there. It was with the newly prepared phosphotungstic acid hematoxylin stains, however, that the true nature of the parenchymal tumor cell became apparent. There were observed a multitude of glial fibrils (fig. 2*C*), most of which stained only indifferently well but many somewhat sharply (fig. 2*D*); the fibrils varied considerably in coarseness, and the finer ones usually stained poorly; none bore any relation to the vessels nor arose from apparent mesodermal elements. That the indifferent staining of fibrils may have been due to immaturity is suggested by figure 2 *E* and *F*, which shows absence of selective staining of fibrils with Hortega's silver lithium carbonate stain. It is therefore apparent that, despite the macroscopic appearance and the initial histologic diagnosis, the diagnosis should be glioma of the optic nerve. Using the Bailey and Cushing terminology for cerebral gliomas, this tumor approached nearest in histologic type the spongioblastoma polare but was atypical in more ways than one (possibly because of its peripheral origin).

The inferential use of "cytoid bodies (Verhoeff[6])" to determine a glial diagnosis is entirely untenable and is being separately dealt with in a publication. Those bodies are found in pituitary adenomas, chordomas, meningiomas, pinealomas, craniopharyngiomas, hemangioblastomas, gummas, the capsules of abscesses and other growths.

5. Verhoeff, F. H., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, p. 1029.

6. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerve, Tr. Sect. Ophth., A. M. A., 1921, p. 146.

ANATOMIC PHORIAS

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COUNCIL BLUFFS, IOWA

I am in accord with the present teachings that phorias may be caused by what is variously termed imbalance of ocular muscles, insufficiency of ocular muscles, lack of enervation and weak sense of fusion. I shall not review these factors but shall call attention to certain anatomic conditions of the eye which are responsible for a phoria of a different type than those usually observed and which require a different kind of treatment.

In a paper on prisms read at a meeting of the American Academy of Ophthalmology and Otolaryngology at Philadelphia in 1921, I presented a table which showed that there were phorias which did not vary in amount over a period of years.[1] Now, fourteen years later, I have again tabulated the results in cases in which I have prescribed prisms. This table presents data on a larger number of cases of exophoria and hyperphoria, and the observations extend over a greater number of years.

In 471 cases of exophoria, the average strength of the first prism prescribed was 1.9 degrees, the average strength of the last prism prescribed was 1.96 degrees and the average time between the first and the last examination was six and ninety-four hundredths years.

In 244 cases of hyperphoria, the average strength of the first prism prescribed was 1.28 degrees, the average strength of the last prism prescribed was 1.26 degrees and the average time between the first and the last examination was five and seventy-nine hundredths years.

I have divided the time the prisms were worn into three periods (from twenty to thirty-five years, from ten to nineteen years and from one to nine years) and have averaged the results in each period, as presented in the accompanying table. Because this type of phoria does not change in amount, I am convinced that it is caused by some unchangeable anatomic condition.

In my experience prisms are not needed in cases of esophoria of the anatomic type. In testing eyes with the Maddox multiple rod one finds that the most common condition is a slight esophoria, which would lead one to the conclusion that a slight anatomic esophoria is a normal condition.

From the Council Bluffs Clinic.

1. Dean, F. W.: Prisms: Should They be Prescribed for Constant Wear? Tr. Am. Acad. Ophth. **26**:108, 1921.

In the study of physiologic optics one is taught that if there is convergence to 3 inches (7.6 cm.) the patient has a reserve power and does not need the aid of prisms. This is not necessarily so.

In examining the eyes of Miss C. R. in May 1935, I found a degree of exophoria which was correctable with a prism of 12 degrees, base in, and yet convergence to 3 inches was possible. There surely was no lack of enervation or of insufficiency of the ocular muscle. In this case the patient could overcome the exophoria of 12 prism degrees and, in addition, convergence to 3 inches was possible. She had a reserve power, and yet her eyes tired and her head ached on doing close work. Because a man may be able to raise a 40 pound (18 Kg.) weight out at arm's length, it does not follow that he can raise and lower a 5 or 10 pound (2.3 or 4.5 Kg.) weight for an hour or so without fatigue.

The anatomic phorias are caused by a malposition of the fovea in relation to its alinement in the cone of the extrinsic muscles. This

Data on Cases of Exophoria and Hyperphoria in Which Prisms Were Prescribed

Type of Phoria	Number of Cases	Period of Years	Average Strength of Prism		Average Period of Years Prism Was Worn
			At First Examination, Degrees	At Last Examination, Degrees	
Exophoria	22	20-35	1.86	1.66	24.72
Exophoria	95	10-19	1.94	2.25	13.0
Exophoria	354	1-9	1.88	1.89	4.47
Hyperphoria	8	20-35	1.93	1.79	24.35
Hyperphoria	43	10-19	1.33	1.33	12.6
Hyperphoria	193	1-9	1.24	1.22	3.56

may be brought about by a slight variation in the position of the fovea in the retina or by a faulty position of the eyeball in the orbit, thus throwing the fovea out of alinement in the cone of the extrinsic muscles when the muscles are at rest.

The fovea need be only slightly misplaced to cause a troublesome phoria. For example, in a case in which the phoria could be corrected with a prism of 4 degrees, the fovea would be misplaced only 0.372 mm. An arc of 1 degree on a circle with a 6 meter radius equals 104.72 mm. Four degrees on that circle would equal 418.88 mm. But the actual deviation caused by a prism of 1 degree is 0.533 degree; so the actual deviation caused by a prism of 4 degrees on a circle with a 6 meter radius is 223.26 mm. The distance from the center of rotation within the eye to the fovea is 10 mm.

From these figures the ratio presented in figure 1 is obtained. Therefore, the fovea would have to be out of alinement in the cone of the extrinsic muscles only a trifle more than 0.33 mm. to result in a phoria which could be corrected with a prism of 4 degrees.

In testing eyes for the position of the fovea in relation to the edge of the blindspot, a tangent screen was used at a distance of 1 meter. It was found that the distance from the blindspot to the fovea varied in different persons and often in the two eyes of the same person. The distance varied from 20 to 23.5 cm., or from 200 to 235 mm., on the tangent screen.

To obtain the actual distance on the retina, the ratio presented in figure 2 was worked out in the following manner: One meter, or 1,000 mm., is to 17 mm., the distance from the focal point in the eye to the fovea, as the distance on the screen is to the distance from the blindspot to the fovea.

In the cases in which examination was carried out the actual distance of the fovea from the blindspot varied from 3.4 to 3.995 mm.

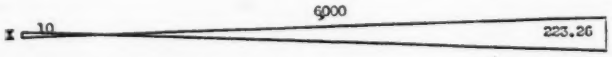

Fig. 1.—Ratio showing the relationship of the position of the fovea to the degree of phoria: 6,000 : 10 : : 223.26 : × = 0.372 mm.

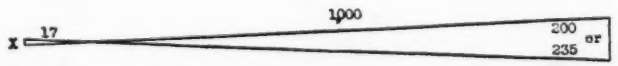

Fig. 2.—Ratios showing the actual distance from the blindspot to the fovea: 1,000 : 17 : : 200 : × = 3.4 mm. 1,000 : 17 : : 235 : × = 3.995 mm. There is a variation of 0.595 mm. in the results.

The foregoing figures show that the relative positions of the fovea and the blindspot vary laterally. They do not prove that the fovea is misplaced in the retina, but they indicate that such a condition is possible, because the distance is not constant. With the present facilities that are available, it is impossible to determine whether the fovea varies vertically in the retina or not, but for some reason the fovea is out of alinement in its relation to the cone of the extrinsic muscles, thus causing hyperphoria. It may be an indication that the eyeball is wrongly set in the orbit.

In going over the records on refraction in the clinic, I found that for 1,020 patients prisms have been prescribed for the correction of hyperphoria. Of that number, 431 had right hyperphoria and 589 left hyperphoria, making a total of 1,020 cases of hyperphoria in 18,440 persons for whom glasses were prescribed. Undoubtedly many cases of hyperphoria have been missed in the examinations, so that these figures must not be taken as a correct proportion of cases of hyperphoria to the number of patients examined.

Whether the malposition of the fovea in its relation to its alinement in the cone of the extrinsic muscles is due to its position in the retina or to the position of the globe in the orbit, the correction is brought about by the same procedure.

One can do nothing to change the position of the fovea in the retina; so in practice one prescribes prisms for constant wear if the phoria is less than 6 prism degrees and an operation on the muscle if it is more than 6 prism degrees.

There are exceptions to this rule. If a patient has a phoria of more than 6 prism diopters and refuses an operation, a prism of 6 degrees is divided between the two eyes, with the hope that this amount of correction may enable the patient to use the eyes without too much discomfort. And again, a young person can overcome a considerable degree of phoria without discomfort. In that case prisms may not be prescribed, but the patient is told that later in life he will probably need prisms.

Whether prisms are used to bend the rays of light to bring the fovea in proper alinement or the alinement is corrected by an advancement or resection, the cure of the phoria is permanent, giving additional strength to the contention that the condition is anatomic and not due to a lack of enervation.

I am thoroughly convinced that there are anatomic phorias, and I have mentioned two factors which may be the cause: malposition of the fovea in the retina and malposition of the globe in the orbit. I am inclined to think that a malposition of the eyeball is the more likely.

Perhaps, as the eyes were being moved to the front of the head during the process of evolution and the ancestors of man took to the trees, where vision was directed mostly to near objects, the process continued until slight esophoria became a normal condition, aphoria occurred often and slight exophoria or hyperphoria occurred in many instances.

One need not go back a million years. Every one started in embryonic life with eyes at the side of the head. The wonder is that the progress of the forward journey of the eyes is stopped in so nearly the right position.

PRIMARY TUMOR OF THE OPTIC NERVE

WITH REPORT OF A CASE

TULLOS O. COSTON, M.D.
BALTIMORE

Since Hudson's [1] excellent paper tumors of the optic nerve have been divided into two main groups: intraneural tumors or tumors of the nerve stem proper and tumors of the nerve sheaths. Most present-day pathologists consider tumors of the first group to be gliomas. Endotheliomas or meningiomas and the various fibromas comprise the second group. A few instances of sarcoma and angioma have been reported, but they will be omitted in this discussion.

GENERAL CONSIDERATIONS

Incidence.—Primary tumor of the optic nerve occurs infrequently. According to Verhoeff,[2] only 300 cases had been reported prior to 1932. Subsequently, reports of only 3 or 4 cases have appeared in the literature. A survey shows that in the reported cases the tumors may be divided as follows: glioma in 80 per cent of the cases, endothelioma or meningioma in 17 per cent and fibroma in 3 per cent.

Little emphasis has been placed on the incidence of primary tumor of the optic nerve in the two sexes, but Hudson's statistics showed a marked preponderance of cases in females. In his collected cases the distribution between the two sexes was as follows: glioma, 70 females and 43 males; endothelioma, 20 females and 7 males, and fibroma, 5 females and 1 male. There are no adequate data on racial incidence.

There is a striking difference in the age incidence of these types of tumors. From 60 to 75 per cent of the cases of glioma occur in the first decade of life, many before the age of 5 years, and from 85 to 90 per cent before the age of 20. In the few reported cases of fibroma a predilection is shown for the early years of life, 4 of Hudson's cases having occurred before the age of 8 years and the other 2 cases before

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Read before the Baltimore City Medical Society, Section on Ophthalmology, Oct. 24, 1935.

From the Wilmer Ophthalmological Institute of the Johns Hopkins University School of Medicine and the Johns Hopkins Hospital.

1. Hudson, A. C.: Primary Tumors of the Optic Nerve, Roy. London Ophth. Hosp. Rep. **18**:317, 1912.

2. Verhoeff, F. H.: Tumors of the Optic Nerve, in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1029.

the age of 32 years. Endotheliomas, on the other hand, usually occur after the first decade, with slightly more than one half of the cases occurring after the age of 30.

Origin and Growth.—By far the greater percentage of gliomas of the optic nerve seem to arise from the intra-orbital portion of the nerve. There are only a few reports of glioma arising from the chiasm or the prechiasmal portion of the nerve. However, as Martin and Cushing [3] pointed out, in a great many of the reported cases of intra-orbital glioma, removal of the tumor was incomplete (in 50 per cent of the cases, according to Hudson's statistics), and the growth may have had an intracranial origin with extension to the orbit. One finds the same uncertainty in determining the origin of the endotheliomas. In many of the recorded cases, removal of the orbital lesion was incomplete, and therefore the point of origin was obscure. Verhoeff [2] has studied eight cases of endothelioma histologically and a number of cases clinically and has been unable to demonstrate an intra-orbital origin. However, there is no reason that the tumor may not arise in the orbit, because the meningeal sheaths about the optic nerve do not differ from those about the brain. The reported cases of fibroma are too few to permit any conclusions to be drawn but in most of them the tumor appears to have had an intra-orbital origin.

In the vast majority of cases a tumor of the optic nerve grows slowly, even for years, but instances of fairly rapid growth during the course of several months are not rare.

Such a tumor does not metastasize. A glioma advances by direct extension along the intraneural portion of the nerve and does not invade the dura. While extension to the chiasm and deeper structures of the brain may occur fairly often, invasion of the disk has been reported in only 4 or 5 instances. An endothelioma or fibroma rarely extends beyond the dura to involve the orbital tissues. While an endothelioma may extend centrally, this type of tumor shows a pronounced tendency to invade the globe. Eight tumors of this type are noted in Hudson's 29 cases, and the following structures are involved: the choroid only, in 4 cases; the choroid and sclera, in 3 cases, and the optic disk, in 1 case.

Diagnosis.—Proptosis and slowly progressive loss of vision are the most common manifestations of an intra-orbital tumor of the optic nerve. Exceptions are often noted, but impairment of vision usually precedes proptosis in cases of glioma, while the reverse is usually true in cases of endothelioma or fibroma. Owing to the fact that a tumor of the optic nerve usually grows very slowly, visual disturbances may begin many years before the onset of proptosis. The direction of the displacement

3. Martin, P., and Cushing, H.: Primary Gliomas of the Chiasm and Optic Nerves in Their Intracranial Portion, Arch. Ophth. **52**:209, 1923.

of the globe usually coincides with the direction of the orbital axis, but exceptions are noted if the growth becomes large.

Limitation of motion of the globe may or may not be present, but it is more often noted in cases of endothelioma and fibroma than in those of glioma.

The most common abnormality found on ophthalmoscopic examination is secondary atrophy of the optic nerve. Papilledema may occur with or without evidence of atrophy and is most frequently observed in association with a growth lying close to the globe or in cases in which intracranial pressure is increased.

One occasionally observes a normal fundus in the presence of an intra-orbital tumor of the optic nerve. In 3 of the 153 cases collected by Hudson [1] the tumor was of this type, and in all 3 it was glioma. In slightly more than one half of his cases atrophy of the optic nerve was shown, and the terms optic neuritis and papilledema were used in describing the condition in the remainder.

In the cases in which the tumor is confined to the intracranial portion of the nerve, diminution of vision associated with atrophy of the optic nerve may be the only findings. If the lesion involves the chiasm these manifestations are bilateral and evidences of a lesion in the chiasmal area may be present, depending on the size and location of the growth. Martin and Cushing [3] found a defect in the temporal portion of the visual field in 3 of their cases. Roentgenographic evidence of enlargement of one or both optic foramina is a valuable aid in diagnosis. The absence of such evidence is no proof that the lesion may not have extended through the foramen, because in many instances such an extension occurs without deformation of the canal.

Papilledema is not produced by a tumor confined to the chiasmal or prechiasmal portion of the optic nerve, unless the growth becomes large enough to cause an increase in the intracranial pressure. Dandy [4] stated that in a large series of cases of intracranial tumor producing primary atrophy of the optic nerve from direct pressure on the optic nerve or tracts, he has not observed papilledema unless the lesion was large enough to cause increased intracranial pressure.

Treatment.—Practically all the authors agree that surgical removal of an intra-orbital growth is the method of choice, unless contraindicated by the patient's general condition.

Before the types of operation employed are discussed, the importance of trying to determine the presence or absence of intracranial extension of an orbital growth should be emphasized. If such an extension exists it is far better to remove the entire tumor by performing a craniotomy and removing the bony roof of the orbit (Martin and Cushing,[3] Dandy [4]

4. Dandy, W. E.: Prechiasmal Intracranial Tumors of the Optic Nerve, Am. J. Ophth. **5**:169, 1922.

and Heuer [5]). This approach, although more hazardous, offers the greatest chance of complete removal of the lesion. If the tumor involves the globe the surgeon should remove all the mass as far as the globe, leaving the latter for enucleation later. Meningitis develops if craniotomy and enucleation are performed at the same time. If one cannot determine the question of intracranial involvement, as is frequently the case, an exploratory operation on the orbit may be performed. Any growth confined to the orbit should be removed, but if extension centrally is observed and the globe is uninvolved, it is wiser to close the orbital incision and later remove the entire lesion from above. If the globe has been invaded it should be removed together with as much of the intra-orbital tumor as can be obtained by the orbital approach, and the intracranial operation can then be carried out later.

There are various methods of orbital exploration. For the anterior approach to the orbit there are the Krönlein operation and procedures which leave the bony orbit intact. One of the latter methods was described by Benedict.[6] An incision is made parallel to and about 6 mm. above the superior orbital margin. The incision is carried through the periosteum, and this structure is freed from the rim and roof of the orbit. The contents of the orbit can then be exposed by a vertical incision in the reflected periosteum and periorbita.

Another method of approach is by means of a transconjunctival incision medially or laterally, depending on the location of the tumor. External canthotomy is usually performed with the lateral transconjunctival incision.

Still another procedure, which has been used with satisfactory results in this clinic, is a modification of that described by Lagrange.[7] This operation is begun with a curved incision in the skin, parallel to and directly over the external orbital rim. By deepening this incision, severing the external rectus muscle and retracting the globe, an excellent exposure of the contents of the orbit is obtained.

Prognosis.—In cases of intra-orbital primary tumor of the optic nerve the prognosis must be guarded because of the possibility of intracranial extension. Local recurrence in the orbit is extremely rare even after incomplete removal. As shown by the experiences of Martin and Cushing,[3] the prognosis is very poor if the tumor involves the chiasm.

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5. Heuer, G. J.: Surgical Experiences with an Intracranial Approach to Chiasmal Lesions, Arch. Surg. **1**:368 (Sept.) 1920.

6. Benedict, W. L.: Removal of Orbital Tumors, Surg., Gynec. & Obst. **58**: 383 (Feb.) 1934.

7. Lagrange, F.: Traité des tumeurs de l'oeil, de l'orbite et des annexes, Paris, G. Steinheil, 1904, p. 506.

REPORT OF CASE

History.—L. S., a white woman aged 48, was admitted to the Wilmer Ophthalmological Institute on March 20, 1934, in the service of Dr. A. C. Woods. She was discharged on April 30, 1934. The chief complaint was loss of vision and prominence of the left eye. The family and the past history were not significant.

Present Illness.—There were no symptoms referable to the eyes prior to the present illness. The illness began twenty years before admission and three weeks after removal of a benign polyp from the intestine. The first symptom consisted of transient attacks of blindness in the left eye. These attacks came suddenly and disappeared within a few seconds. They gradually became more frequent during the course of a few weeks, and vision in the left eye slowly failed until, at the end

Fig. 1.—Photograph of the fundus, showing extension of the tumor to the temporal side of the disk.

of six months, no perception of light remained. The patient stated that at about this time she thought the left eye appeared more prominent than the right but that the oculist whom she consulted stated that no proptosis existed. Within the next few months, however, proptosis of the left eye became manifest, gradually increased for a year or two and then remained stationary until about six months before admission. During the six months prior to admission the proptosis gradually increased. She experienced no discomfort in the eye except two or three sharp, jabbing pains in the left globe about one week before admission. The right eye had been asymptomatic at all times.

Examination of the Eyes.—Right Eye: The right eye was entirely normal, with the vision 20/15 and a normal field.

Left Eye: External examination revealed proptosis of 8 mm. There were slight fulness and puffiness of the lids, but no tumor could be palpated. The exter-

Fig. 2.—Cross-section of the optic nerve and disk, showing invasion by the tumor.

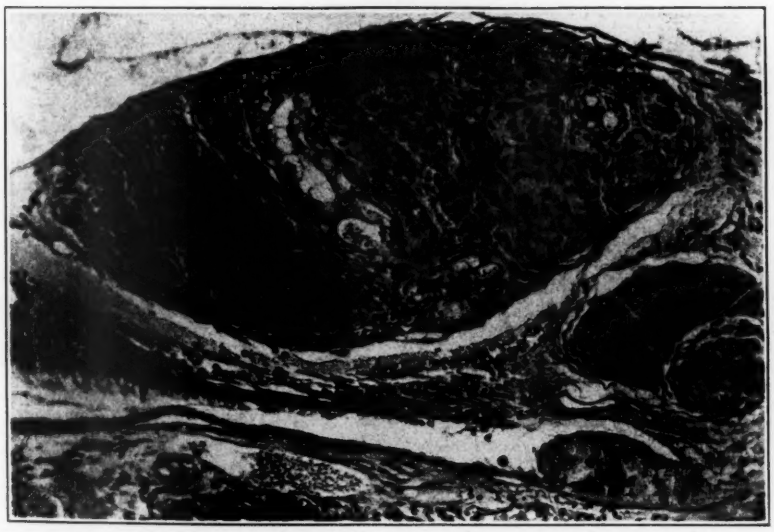


Fig. 3.—High power magnification of the tumor cells on the temporal edge of the disk.

nal ocular muscles were normal except for definite limitation of motion temporally. The conjunctivae, cornea, tension, iris, anterior chamber, pupil and lens were normal. Ophthalmoscopic examination revealed that the media were clear. The disk was elevated approximately from 5 to 6 diopters, and there were marked blurring and edema of the margin associated with generalized pallor. Physiologic cupping was absent. Several dilated capillary loops were seen on the disk. All the arteries were narrow, and the veins were distended. On the temporal edge of the disk there were two elevated, grayish nodules, which appeared to be definitely connected with the swollen disk. One of these was about twice the size of the other and is shown plainly in the photograph of the fundus. The macular area and the peripheral portion of the fundus were entirely normal. Examination with the slit lamp revealed the anterior segment of the globe to be entirely normal. Tests for vision showed no perception of light.

Physical Examination.—A general examination, including that of the nose and throat, revealed no abnormalities. Laboratory tests gave the following results: basal metabolic rate, +10; blood chemistry, normal; red blood cells, 4,190,000; white blood cells, 8,100; hemoglobin content, 82 per cent; clotting time, four and one-half minutes; urine, normal, and Wassermann reaction of the blood, negative. Roentgenograms of the entire head, including the sinuses and optic foramina, showed nothing abnormal.

Operation.—Operation was performed on March 22, 1934, by Dr. A. C. Woods. Through a modified Lagrange incision a firm mass could be palpated, and it was observed to surround the optic nerve completely from the globe to the apex of the orbit. The tumor apparently stopped short of the optic foramen. The globe, the tumor and the intra-orbital portion of the optic nerve were excised. The operator was fairly certain that all the tumor had been removed. The tumor appeared to be entirely encapsulated and had not extended into the tissues of the orbit. Convalescence from operation was uncomplicated except for a mild purulent infection, which remained entirely localized in the orbit and subsided after a few weeks.

The patient was entirely well when last heard from one year after operation.

Pathologic Report (Dr. Jonas Friedenwald).—The anterior portions of the eye were normal. Immediately behind the optic disk a fusiform tumor was observed, which measured 12 by 12 by 15 mm. in diameter and which completely surrounded the optic nerve. For the most part the tumor was surrounded by the dural coat of the optic nerve sheath, but it had broken through the dura at a few points and had also extended through the optic disk into the retina. Even at these places, however, the tumor was surrounded by a delicate capsule. It was composed of irregular lobules, separated from one another by dense fibrous trabeculae, the lobules consisting of syncytial masses of large cells with pale-staining vesicular nuclei and homogeneous protoplasm. No mitotic figures were seen. The optic nerve had been compressed by the tumor into a narrow cord and was completely atrophic. There was slight papilledema.

Diagnosis.—The diagnosis was dural endothelioma of the optic nerve, with invasion of the optic disk and atrophy.

Comment.—The case reported here is unusual because the extension of the growth to the disk was recognized ophthalmoscopically and photographed and the diagnosis was subsequently proved by histologic examination.

Clinical Notes

THE PRECISION ANGLOMETER

GEORGE E. PARK, M.D., CHICAGO

The goal in operation for concomitant squint or in orthoptic training should be to establish balance of the extra-ocular muscles so that when the visual axes are fixed at infinity there is no excessive pull on any muscle or muscles in order to maintain fusion.

To date there have been only a limited number of instruments worthy of consideration which are sufficiently accurate for making such measurements of the different angles of reflection and deviation as will furnish basic facts on which laws can be established. The Stevens tropometer is the only instrument which has been used to determine the relative strengths of the individual rectus muscles. In this instrument a tangent scale was used, which could be set at variable distances from the eye to be measured. This necessarily gave faulty readings by reason of the varying distances of the scale from the eye.

It is believed that the data which can be obtained with the use of the instrument to be described in this paper will eventually make possible a gaged and relatively exact operation in cases of concomitant squint. Not only will it be possible to determine the relative length, strength and leverage of the external and internal rectus muscles in order to know the exact amount of advancement or recession which is required to give the eyes perfect balance, but a large amount of other important information is made available by the instrument, which will be mentioned as the technic of its use is described.

This instrument,[1] which is in use at present in the department of ophthalmology of the Northwestern University Medical School, was designed and constructed by Mr. Clile C. Allen to embody the results of research conducted by Dr. Russell Smith Park, professor and head of the department of mathematics, Eastern Kentucky Teachers' College,[2] and me.

From the Department of Ophthalmology, Northwestern University Medical School.

Presented at the Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Cincinnati, Sept. 17, 1935.

1. For forty years Mr. Allen has been a student of optics and mechanics, and as a result of his unusual ingeniousness, he has been able to incorporate his knowledge into this instrument. He has done this work purely for the advancement of science and without any thought of financial gain.

2. Park, R. S., and Park, G. E.: Center of Ocular Rotation in Horizontal Plane, Am. J. Physiol. **104**:545 (June) 1933.

THEORETICAL CONSIDERATIONS

The research which my associate and I carried on disclosed a point within the orbit that is fixed with relation to the head but not with relation to the eye, through which the visual axis passes during all horizontal excursions of the eye. We have termed this point, which lies within the eye, the visual center. We also found that the kinematic center of ocular rotation moves along a curve at a variable distance of from 1.066 to 0.893 mm. nasalward from the visual axis and always on a line perpendicular to the visual axis at the visual center. Also there are variable distances of 14.732, 13.918 and 12.95 mm. from the point where the visual axis passes through the anterior surface of the cornea to the visual center as the angle of the visual axis takes the positions of 39 degrees and 4 degrees on the nasal side, and 38 degrees on the temporal side, respectively.

As the visual axis swings on this point as a center during an excursion of the eye, it logically follows that the angular amount of ocular movement should be measured with reference to this center.

The importance of the visual center is evident in another respect, viz., that the visual axis seldom intersects the center of the pupil. This being true, it is evident that any difference between the intercentric distance of the two visual centers and the interpupillary distance results in an effect which resembles that of a prism when no prism is prescribed, owing to decentration, or when a prism is prescribed, the resultant power of the prism will be increased or decreased, the strength and direction of the base of such a prism in either case being dependent on the strength and sign of the prescribed lenses.

Greater accuracy is attained by prescribing the intercentric distance of the visual centers for the centering of lenses instead of the usual interpupillary distance.

THE INSTRUMENT

The instrument, as shown in figure 1, is so constructed as to embody the features described. It consists of a head support designed to hold the head firmly in a fixed position, facing which is a telescope so arranged that its axis may be brought to coincide with the visual axis of the eye. For this purpose the telescope carries an illuminated target (fig. 2), which the patient views in establishing this coincidence of the axes. The telescope is movable along a graduated arc in such a manner that the vertical projection of the center of the arc always intersects the axis of the telescope. Means are provided for bringing this point of intersection to coincide with the visual center of the eye so that all angular measurements of the movements of the telescope along the arc are made with reference to the visual center.

Means are also provided for determining the separation of the visual centers.

TECHNIC OF USE OF THE INSTRUMENT

Fixation.—The patient is asked to bite a removable bit of wood (*1*), which is held in slots in the head support by the screw *2*, while elastic pads are brought into contact with the forehead, temples and back of the head by their respective screws (*3*, *4*, *5* and *6*). By this means the head is held firmly in a fixed position.

Primary Position.—The telescope is then brought to the zero position on the arc by the knurl *7*. Next the telescope is adjusted before the eye horizontally by the knurl *8* and vertically by the knurl *9*, until the patient sees a bright spoked wheel,

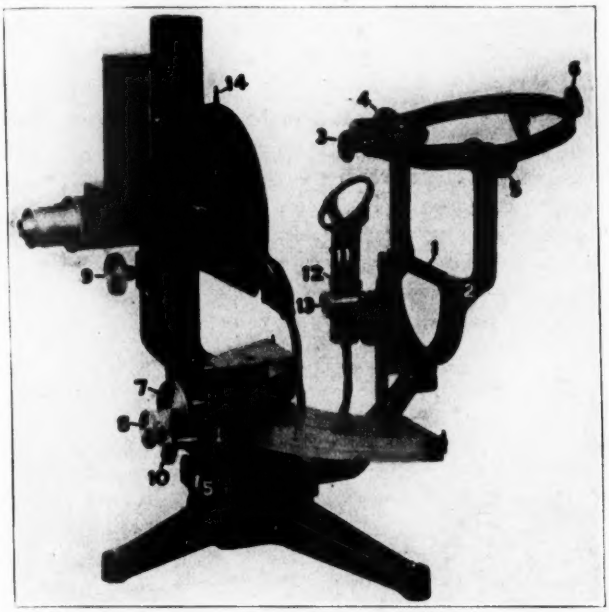


Fig. 1.—Photograph of the precision anglometer.

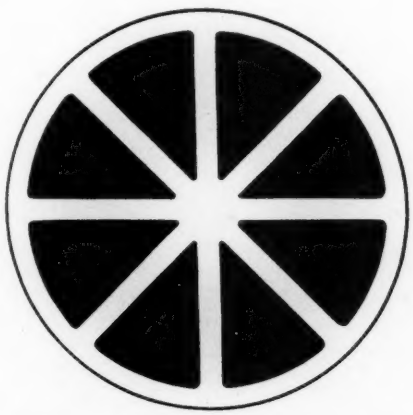


Fig. 2.—Illuminated target, measuring 9/16 inch (1.4 cm.), used in the telescope of the precision anglometer.

the target, at the center of a dark circle (fig. 3*A*), as shown in figure 3*B*. The rim of the wheel is slightly smaller than the dark circle, and the patient easily sees when the wheel fits centrally into the dark circle. When this is done the visual axis coincides with the axis of the telescope.

Next the telescope is moved by the knurl *10* toward or from the eye, which is looking through the eyepiece, until the image of the target, reflected from the cornea, is seen focused on the scale of the eyepiece (fig. 4). When this is done the visual center of the eye coincides with the intersection of the vertically projected center of the arc and the axis of the telescope, and all angular measurements of movements of the eyes are made with reference to the visual center.

The image of the target does not usually lie at the intersection of the central horizontal and vertical lines of the scale of the eyepiece, as the axis of the surface of the cornea usually lies at an angle to the visual axis. This angle causes an oblique reflection of the target from the corneal surface, and its image seen at the scale of the eyepiece usually lies below and at one side of the center of the scale and is astigmatically distorted. There is always one meridian of the wheel, however, which can be focused in conjunction with the scale of the eyepiece. This angle between the axis of the cornea, which is the optical axis of the eye, and the visual axis is the physiologic angle. The term physiologic angle is used here

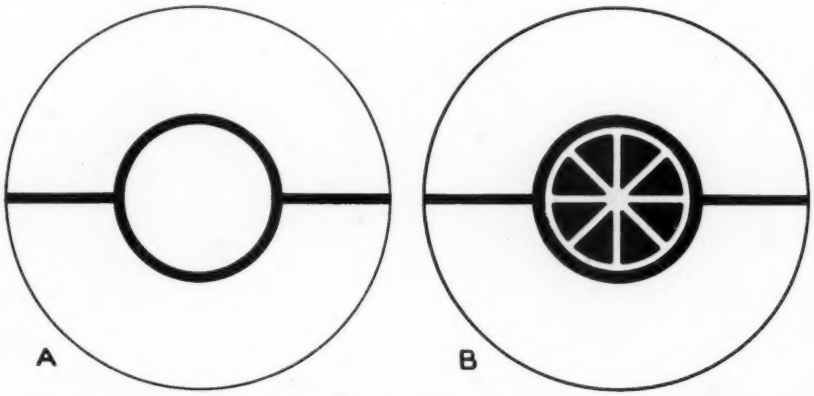


Fig. 3.—Drawing showing (*A*) the heavy black circle, measuring 1½ inches (3.8 cm.) in diameter, on which the target is centered and (*B*) the target centered on the dark circle.

instead of the confused nomenclature alpha, gamma or kappa angle of Landoldt, since there is but one angle to be considered. Reasons for this will be given in a later publication.

Determination of the Physiologic Angle.—The instrument is set in the primary position; the light in tube *11* is switched on, and its transparent mirror is adjusted before the eye vertically by the knurl *12* and horizontally by the knurl *13*, until the patient sees a small red spot superimposed on the center of the target. While the patient is directed to fix on this red spot, the telescope is moved along the arc by the knurl *7*, until the center of the target is brought on the central vertical line of the scale of the eyepiece. The angle indicated on the arc is the physiologic angle, as the optical axis of the eye coincides with the axis of the telescope while the visual axis is held at zero by fixation on the red spot.

Separation of the Visual Centers.—The exact determination of this separation is necessary for optical measurements only when there is vision in both eyes. The instrument is set in the primary position before one eye, and the scale (*15*) is

moved to indicate the zero position for this eye. The telescope is moved before the other eye by the knurl *8*, and the primary position for that eye is secured. When this is done the scale indicates the separation of the visual centers.

When vision is present in but one eye, it is sufficient to use the scale (*15*) in conjunction with the method for securing alinement of the blind eye, to be described in paragraphs 2 and 3 of the following section, or with any other method, as the separation of the lenses is a matter of cosmetics and separation of the visual centers is not important.

Determination of Strabismus.—1. Cases in Which Vision is Present in Both Eyes: The instrument is set in the primary position before one eye, and the tube (*11*) is adjusted to the point at which the red spot is seen at the center of the

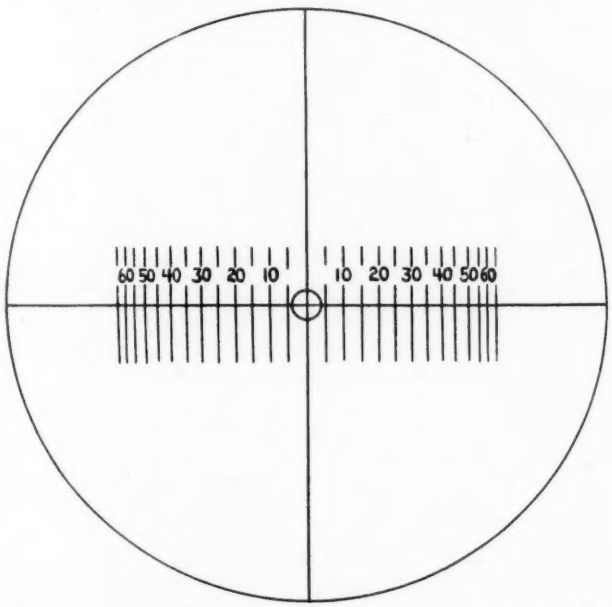


Fig. 4.—Scale of the eyepiece, measuring 1½ inches (3.8 cm.) in diameter.

target. Next the telescope is brought before the other eye by the knurl *8*, and the knurls *8* and *9* are adjusted for the primary position before this eye.

The patient is directed to fix the red spot again and move the telescope along the arc by the knurl *7* until the red spot is again seen at the center of the target. The first eye is now fixing the red spot, and the second, the target. As these two objects are dissimilar, there is no tendency to fusion, and the angle indicated on the arc is the visual angle of the deviation.

If it is found unnecessary to move the telescope from zero on the arc to center the red spot on the target, it is known that orthophoria is present at infinity. But if heterotropia or heterophoria is present, the red spot is not superimposed on the target and must be brought to superimposition by the knurl *7* to determine the angular amount of the disturbance, as indicated on the arc. Means are not provided for measuring hyperphoria or hypertropia.

2. Cases in Which Vision is Present in But One Eye: The instrument is set in the primary position before the normal eye and the tube (*11*) is adjusted to the point at which the red spot is seen at the center of the wheel. The telescope is brought before the other eye, and the patient is directed to look to one side in the direction necessary to bring the observed eye approximately into alinement with the telescope. By slight variations in the direction of the patient's gaze and by adjustment of the knurls *7* and *8*, image of the target reflected from the cornea is brought to the same distance from the central vertical line of the scale of the eyepiece at which it appeared for the first eye but on the opposite side of the line. The patient is directed to look again at the red spot and to move the telescope along the arc by the knurl *7* until the image reflected from the cornea is again seen in the position just described. The angle indicated on the arc is the angle of deviation.

3. Cases in Which the Pupil of the Deviating Eye Lies Under a Canthus: The next procedure is that just described for the normal eye and for the alinement of the second eye with the telescope. In cases in which this alinement cannot be effected the practitioner must use his judgment in adjusting the telescope before the deviating eye. After the telescope has been brought in this position, the position of the portion of the corneal margin opposite the canthus is noted on the scale of the eyepiece, and the patient is directed to look again at the red spot. The position is noted to which the portion of the margin of the cornea has moved across the scale. The angle of deviation is the sum of the angles noted on each side of the center of the scale.

The graduations of the scale must not be confused with those of the arc, as the graduations of the scale are used only in cases of the condition just described and in all other observations those of the arc are used.

Determination of the Strength of the Rectus Muscles.—The instrument is set in the primary position, and the light in the tube (*11*) is switched off and moved out of the way. The eye orbit is illuminated by moving the bar (*14*) to the right. The patient is directed to fix the center of the target and to move the telescope along the arc by means of the knurl *7* until an angle is reached at which the patient can no longer steadily fix the target. The correct angle is easily found by observing through the telescope where the eye begins to waver and can fix but momentarily. This gives the rotating power of the individual muscle in terms of degrees of the arc, from which information is obtained as to the relative length, strength and leverage of the muscle. If the pupil of the deviating eye lies under a canthus after the rotation, the measurements are made as described in paragraph 3 of the preceding section.

CONCLUSIONS

An instrument is described which, it is believed, allows for greater accuracy in measuring the ocular movements than is possible with previous instruments.

This accuracy depends on the fact that the axis of the telescope coincides with the visual axis, and the center of its arc of rotation with what we have termed the visual center of the eye. This is brought about by the construction of the instrument and by an accurate device to insure fixation at all times. It is of interest that the telescope of Gullstrand [3] also incorporates a target which is reflected from the cornea

3. Gullstrand, Allvar: Photographisch-ophthalmometrische Untersuchungen über die Hornhautrefraction, Stockholm, P. A. Norsted & Söner, 1896.

for the sole purpose of observing irregularities of the surface of the cornea but that it is not provided with means for measuring angular movements of the eye.

Aside from its advantages for scientific investigation of the ocular movements, our instrument is adapted for clinical use. After one has become familiar with its use, a complete record of the horizontal movements may be made in from five to ten minutes. Measurements of the vertical movements require the use of a more complicated instrument than is considered practical at this time.

A NEW KERATOSCOPE

WILLIAM H. HOWARD, M.D., CHICAGO

The Placido disk is acknowledged to be one of the standard units of the modern ophthalmologic clinic. Every student of the eye soon becomes acquainted with this unique device, if not from practical use at least from the pages of his textbook. One can safely state, however, that most clinics use it for a very limited scope of their activities.

Certain European investigators, notably, Amsler,[1] of Lucerne, Hartinger,[2] of Jena, and Fischer,[3] of Leipzig, have felt the need of improving on the Placido disk; so there are electrically lighted disks, which require a camera for photographing the image reflected from the cornea. The principle used in the designing of these photokeratoscopes is found in most ophthalmoscopic instruments; namely, precision is effected by contrasting light with a dark background. These European instruments have proved to be an added refinement in the study of the irregular cornea and in the diagnosis of attending abnormalities. However, there are certain disadvantages to be considered. Their initial cost and the expense of frequent use are definite items. The time required for their operation and the other obvious qualifications for good photography are also important. Large instruments are necessary for stationary use only. These factors discourage the frequent use of these complicated instruments; at best, they lend themselves almost exclusively to the work of private investigators.

Presented at the American Academy of Ophthalmology and Oto-Laryngology, Cincinnati, Sept. 17, 1935.

1. Amsler, M.: Ueber Photo-Keratoskopie, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **48**:202, 1930.

2. Hartinger, H.: Ein neues Photo-Keratoskop, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **48**:360, 1930; Ueber einen neuen Corneal-Reflektographen, ibid. **49**:495, 1932.

3. Fischer, F. P.: Vorweisung von Reflexphotographien, hergestellt mit dem neuen Zeissschen Corneal-Reflektographen, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **49**:499, 1932.

Fig. 1.—Front view of the instrument.

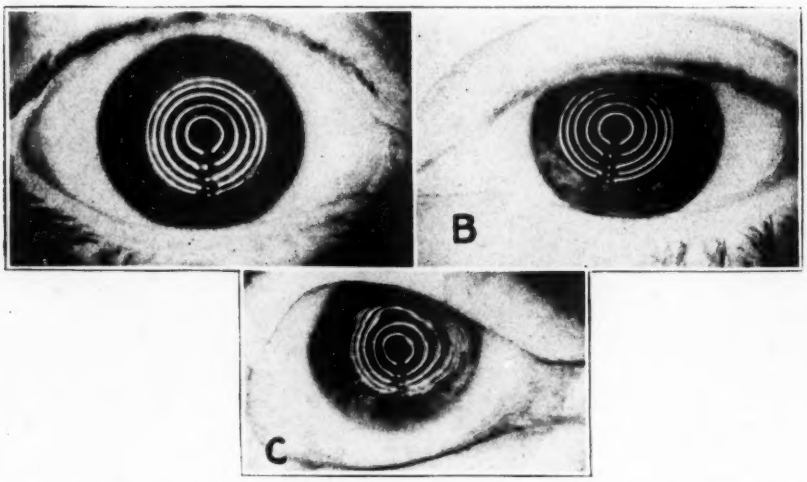


Fig. 2.—Photographs showing appearance with the keratoscope of (*A*) the normal cornea with a slight defect in the lower nasal portion, which is frequently found in otherwise normal eyes, (*B*) a cornea presenting regular astigmatism, requiring a correction of —2.00 sph. +5.50 cyl., axis 105° and (*C*) a cornea showing irregular astigmatism in a case of old trachoma with healed ulceration of the cornea.

Obviously, a keratoscope is not required for use as a routine, but when it is needed such an instrument should be easily available and handy to use and should not be of prohibitive cost.

A newly designed keratoscope, which allows direct examination of the eye without the necessary use of a camera, is presented.

The instrument is a black disk, measuring 10 inches (25.4 cm.), with a central aperture and five concentric grooved rings, patterned to fit the special tubing, which contains neon and has a positive and a negative electrode. It is arranged to give the greatest possible symmetry to rings which must necessarily be incomplete. The peculiar design at the position of 6 o'clock is the result of painting out the bights of each circle and likewise the segments of the perpendicular tube, which leads to the first central ring. A hollow hard rubber handle supports the disk and encloses the two electric cords leading to the small transformer, which operates on an alternating current. (The original model prepared for manual use can easily be constructed to fit a sliding platform, thereby giving double service.) A central sleeve projection contains a magnifying lens, with a posterior catch to allow the insertion of either a minus or a plus lens. Special diagrammed charts to record the findings may form a part of the complete equipment. In the exceptional case in which a photographic copy is wanted, for example in legal proceedings, case reports, etc., a camera can be used.

The principal advantage of this instrument is the opportunity for a clearly defined, direct, simple and easily repeated examination of the cornea under almost all conditions. Its use is indicated in the examination of the cornea of irregular topography, either of congenital origin or acquired owing to disease, trauma or operation. It can be used for class demonstration in defining the normal cornea.

9120 Commercial Avenue.

A NEW ELECTRODE FOR SURGICAL DIATHERMY OF THE RETINA

CLINTON T. COOKE, M.D., PORTLAND, ORE.

For some time it has seemed to me that the operation for detachment of the retina should be simplified. The multiplicity of micropins used in the methods of Walker and Šafář has necessitated a technic which is so complicated and difficult that most ophthalmologists have hesitated about attempting to perform the operation and have been forced to choose between giving a patient unskilled or inexperienced service or referring him to some metropolitan wizard.

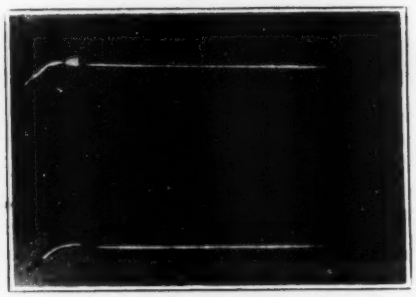
For several months I have been using with ease and satisfaction to myself and to my patients an electrode of my own design. The electrode consists of an iridioplatinum pin, 0.3 mm. in diameter, mounted on a silver point, which forms a shoulder, 1 mm. in diameter. This in turn is screwed into the end of a tapered vulcanite handle, about the

size of a fountain pen, 14 cm. long and about 6 mm. in diameter. An electrical conductor in the long axis of the handle is attached to a very flexible gum-covered wire cord that connects with a diathermy current.

Any source of surgical diathermy current may be used. I have used both the Walker and the Majestic unit.

There are three interchangeable silver and iridioplatinum points; one point can be removed and another quickly substituted during an operation. The shortest micropin is 1 mm. long and is designed for use under the muscles and in front of the equator of the eyeball. The next sized point is 1.5 mm. long and is used back of the equator. The third point is used for puncturing to effect drainage of subretinal fluid and is 2.5 mm. long and 0.5 mm. in diameter.

It will be seen that with this simple electrode one can make any number of scleral punctures desired in one, two, three order nearly as fast as the foot switch can be stepped on. The shortest pin penetrates to the choroid and sets up controllable adhesive choroiditis, which holds the retina in situ after drainage of the subretinal fluid through the punc-



New electrode tips for use in operations for detachment of the retina.

tures made by the larger and longer pin. If desired, the pin, being of iridioplatinum, can be freed of any coagulum that may adhere to it by passing it through a flame or by wiping it with gauze.

It is possible to make the punctures under a muscle in any desired line without dividing the muscle, simply by holding it aside with a pair of bridle sutures, with an Ainslie nonconducting retractor or with a strabismus hook over which a length of a very fine rubber tubing has been drawn.

The time of performance of the operation is shortened to a few minutes, and any operator who is able to do an operation for strabismus can perform this operation with very little trauma to the eyeball.

The location of the holes in the retina may seem desirable to some surgeons, but I do not feel that it is absolutely necessary. In the cases in which I have not been able to discover any hole the patients have done as well as others in whom the hole was located.

After I had begun using this device I was interested to read Dr. Gradle's description of his electrode and of his experience with it, which confirms what I have said here.

This new electrode may be obtained from the Shaw Supply Company, of Portland, Ore., and Seattle, Tacoma and Spokane, Wash.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

EVOLUTION OF PERIMETRY

RALPH I. LLOYD, M.D.

BROOKLYN

The early Greeks and Romans knew that the visual fields had definite limits, but one finds no exact figures prior to those of Venturi, who gives the vertical extent as 112° and the lateral as 135°. The part in the books on optics written by Ptolemy (150 A. D.) containing the data given in the introductory quotation from Arago's work has been lost, and more exact information about his ideas on this phase of optics is not available.

Euclid taught that a fluid streamed from the eye in the form of a cone with the point in the pupil and the base on the object looked at; in other words, the base of the cone was the visual field. Helidore of Larisse described the base of this cone as circular. All inside this base was visible and all outside invisible.

Astronomers and philosophers of all ages have made important contributions to optics, and the first group discovered very early that the macular area is nightblind. They recommended observation of the stars with the paracentral retina and agreed that 11° lateral to the fovea is the point of greatest efficiency. This is in accord with the anatomic arrangement of the rods and cones, for in this zone the ratio of rods to cones reaches the maximum, 20: 1, which is constant thereafter.

Another contribution by nonmedical observers concerns migraine, hemianopia and the arrangement of fibers in the chiasm. Sir Isaac Newton was afflicted with migraine and could explain the hemianopic features of the scintillating scotoma so characteristic of the ailment only by assuming a hemidecussation of the tract. Brewster approached

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Ptolemy announced he had recognized experimentally in the field of vision, that the space visible in an invariable position of the eye is limited by a rectangular cone, that is to say, by a cone with its summit at the pupil with the angles diametrically opposite, perpendicularly between them. This data has been conveyed to us by Helidore of Larisse. It results that in order to see at the same glance of the eye, the horizon and the zenith, it is necessary to direct the visual axis 45° above and that the eye turning in its orbit at the horizon, never lets us see more than a quarter of the surface of the sky.

ARAGO.

the same problem in trying to explain why the sun when observed through a tube by one eye produces a bilateral after-image instead of a unilateral one. In this study he seriously injured his eyes, and they never fully recovered. Wollaston suffered from hemianopic scintillating scotomas after a severe physical effort. Twenty years later, he had a permanent right homonymous hemianopia that caused him to order an autopsy, which revealed a lesion in the left optic thalamus. In 1723, Vater and Heinecks wrote:

> We infer that the optic nerves in that bundle union scatter their fibers and unite them so that after withdrawing therefrom they separate into two equal parts corresponding to the hemispheres of the brain; so that the right side of the coated retina of each eye receives the filaments from the right hemisphere, the left side certainly receives the filament from the left hemisphere, and therefore the eyes feel between them an equal distribution on the coated retina in the process of vision.

Galen seems to have been the first physician to record a recognition of extramacular fields in opposition to the idea that vision is a simple function, alike in all parts of the retina except in degree of efficiency. What is probably the first illustration of a field of vision is shown here (fig. 1). It dates from 1602 and was found in comparatively recent years by Aubert in an article by Ulmus of Padua. This writer said, "We believe the cheek bones, the nose and the forehead have been made naked and unprovided with hair by the divine creative faculty for one end, that is the act of vision." The line *H-I* indicates what he considers to be the field of vision but, he stated, if hair grew on the forehead in the manner shown, or on the cheek, the limits of the usable field would shrink to *F-E-G*.

The first reliable observations on the area of the visual fields and of the zone of acutest vision must be credited to Thomas Young. From his lecture given in 1801 comes the following:

> The visual axis being fixed in any direction, I can at the same time see a luminous object placed laterally at a considerable distance from it, but in various directions the angle is very different. Upwards it extends to 50°, inwards to 60°, downwards to 70° and outwards to 90°. These internal limits of the field of view nearly correspond with the external limits formed by the different parts of the face, when the eye is directed forwards and somewhat downwards, which is the most natural position; although the internal limits are a little more extensive than the external, and both are well calculated for enabling us to perceive the most readily, such objects as are the most likely to concern us. . . . It is well known that the retina advances further forwards toward the inner angle of the eye, than towards the external angle; but upwards and downwards its extent is nearly equal, and is indeed every way greater than the limits of the field of view, even if allowance is made for the refraction of the cornea only. . . . The whole extent of perfect vision is little more than ten degrees; or more

strictly speaking, the imperfection begins within a degree or two of the visual axis, and at the distance of five or six degrees becomes nearly stationary, until, at a still greater distance, vision is wholly extinguished.

Purkinje was the next important investigator of the limits of the visual fields, and he used the methods introduced by Young. In 1825, he found the outer limits at 100°, the inner at 60°, the upper at 60° and the lower at 80°. He found the outer limit increased to 110° when the pupil was dilated. Boerhaave recognized scotomas in 1708, and Beer in 1817 described contractions of the visual field. The medical profession seemingly did not utilize these investigations or those of Hueck

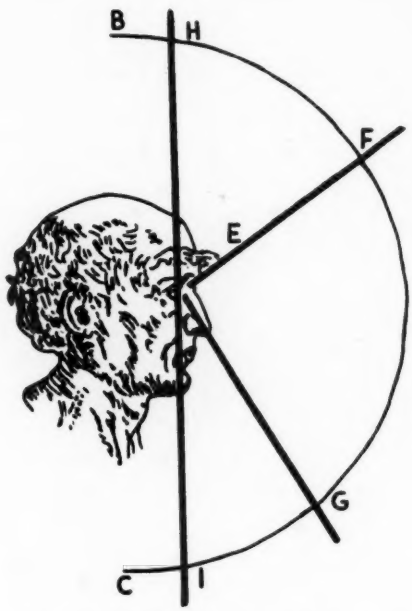

Fig. 1.—Probably the first published illustration of the field of vision (from an article by Ulmus of Padua, 1602). The line *H-I* indicates the field of vision. *F-E-G* shows what the limits of the field would be if hair grew on the forehead or on the cheeks in the manner shown.

and Volckmann, made with parallel lines and pairs of dots as test objects to determine how far from the fixing point they could be recognized as separate objects.

The first reports of actual employment of the results of examinations of the visual fields to aid in diagnosis emanate, like so many other ideas, from von Graefe. In 1855, he published his classic work entitled "Examination of the Visual Functions in Amblyopic Affections." This article is conclusive evidence that he had been making careful examinations of the central and paracentral areas for some time and had carried his tests as far from the fixing point as a flat surface would permit. Hemi-

anopic and sectorial defects, the enlarged blind spots of myopia, ring-scotomas and superior hemianopias found in retinal detachments were all well known to him. The ophthalmologic literature of Germany begins in 1820, and two of the four journals in existence prior to von Graefe's *Archiv für Ophthalmologie* contain no articles on visual fields nor is the subject mentioned in any of the case reports. I have not seen the other two journals, but their publication was very early and did not continue for any great period. It is also evident from von Graefe's article and others following it that examinations of the visual fields against a flat surface were more highly esteemed prior to the invention of the ophthalmoscope than afterward because before 1851, when von Helmholtz made his great contribution to ophthalmology, no direct approach to retinal lesions was possible, and the only way to diagnose intrabulbar and retrobulbar lesions was to imagine the field of vision as laid on a flat surface before the patient, ready for examination. This field must then be searched for defects, and from their character retinitis pigmentosa, glaucoma, retinal detachment and optic atrophy were differentiated from lesions of the brain, optic tracts and chiasm. Who first realized that some test other than that of reading test types was necessary cannot be determined. Von Graefe used a small blackboard at 18 inches (45.7 cm.) from the patient with a small piece of chalk at the end of a wire as the test object. With this simple apparatus he did what only a man of his caliber could but was unable to determine the limits of the field, nor did he go into the use of colors. He also used a surface with letters, figures or dots arranged about the fixing point to enable the patient to recognize the limits of the functioning area more easily. This very practical plan makes the patient aware of the line where recognition ceases. It has recently been reintroduced by Salzer under the name of *Blick-Perimetrie*.

Another suggestion by von Graefe brought into general use the division of the flat surface into numbered squares. Figure 2 shows his mappings in a case of enlarged blind spots and ring scotomas. He stated that homonymous half losses are caused by unilateral cerebral disease and heteronymous hemianopias by growths at the base of the brain which press on the cruciform fibers of the chiasm. In the third volume of his *Archiv*, he reported a scotoma caused by a foreign body (bird shot) entering the eye and glancing from the first contact with the sclera to rest finally in an absolute scotoma opposite. Figure 3 shows the first impact after the bird shot entered the eye at *C*, and the path across the eye, *B*, leading to the final resting place, *A*. *C* and *B* are relative defects, but *A* is absolute. He complained that his colleagues did not make studies of the fields often enough to complete their examinations and improve their diagnoses. Also, he condemned

the use of a candle flame as a test object because of the irradiation. His classic article on glaucoma, which appeared in 1869, shows a familiarity with the effects of increased pressures that is truly amazing, and the average reader will be surprised to find that von Graefe was familiar with the defect known as Roenne's nasal step and the finger-like defects running from the blindspots.

Only the contributions of Donders and his colleague, Haffmans, printed in German, are available to me, and for that reason I am not able to do justice to the work of the Holland school. It is nevertheless

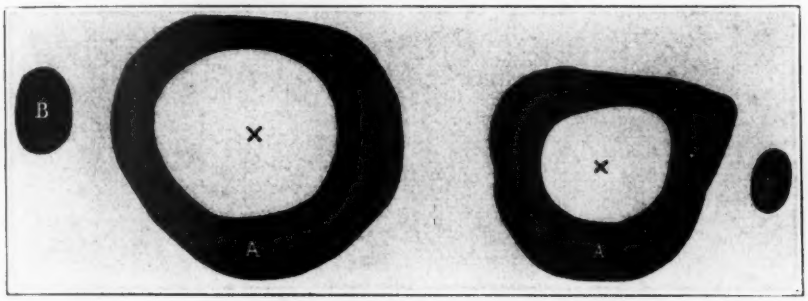

Fig. 2.—Enlarged spots and ring scotomas outlined by von Graefe.

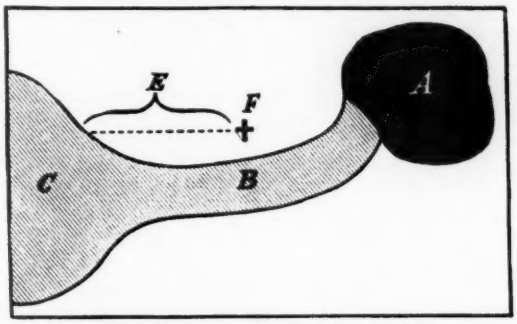

Fig. 3.—Defects outlined by von Graefe. A bird shot entered the eye and struck at the point corresponding to *C*. *B* marks the path to the final resting place at *A*. *F* is the fixation point.

evident that these men were thoroughly familiar with defects resulting from glaucoma as we know those defects today, especially with the early loss of one of the nasal quadrants.

The year after the publication of the first von Graefe classic, Aubert and Foerster began their contributions to detailed study of the functions of the retinal periphery. The first article by this esteemed pair of investigators was entitled "Studies of the Knowledge of Space of the Retina," and this was followed by an article by the first mentioned one alone, entitled "On the Boundaries of the Color Recognition of

the lateral Parts of the Retina." At first, they used the well known flat surface with letters or figures arranged about the fixing point. The test was conducted in a darkened room with the eye protected from the direct rays of the flash of the Reis bottle (fig. 4[1]) by a tube with a black lining. Movement of the eye under examination was avoided by the flash lighting originally introduced by Volckmann. The results

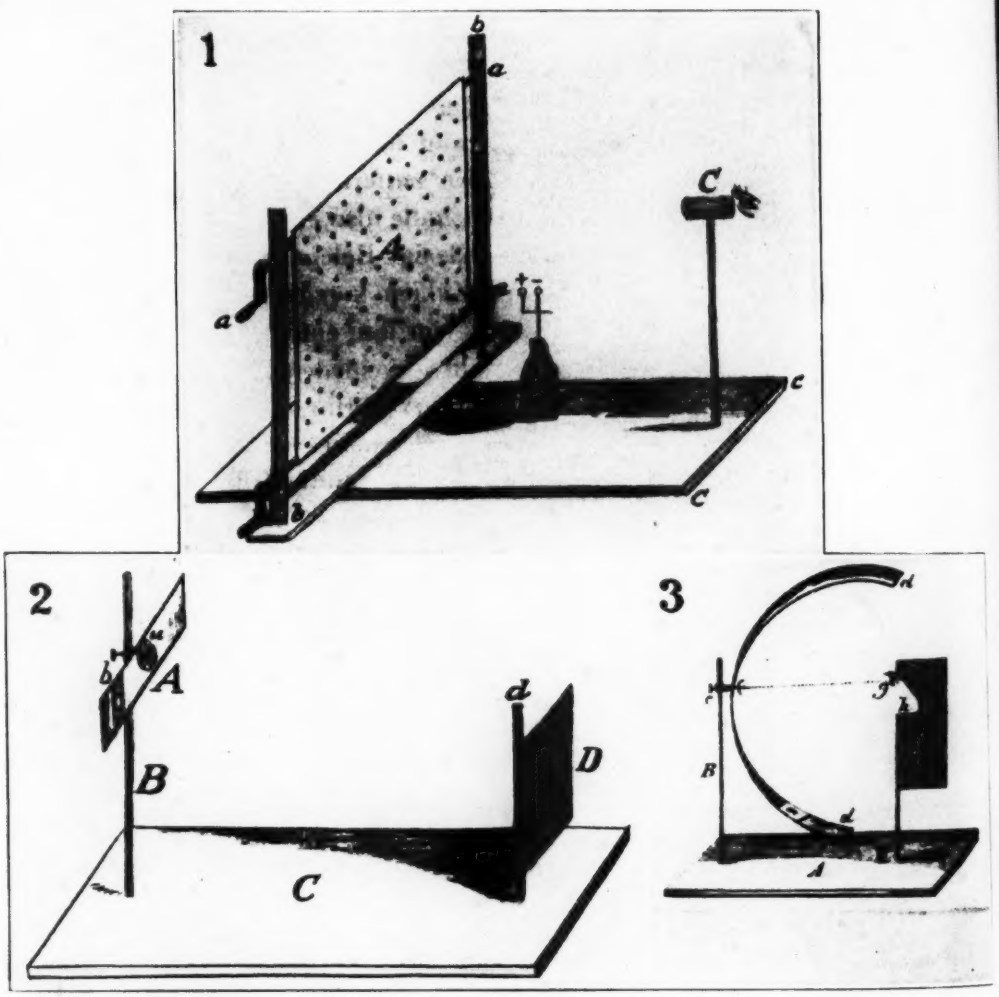

Fig. 4.—*1*, the first campimeter used by Aubert and Foerster. *C* is the tube to protect the eye from the flash of the Reis bottle, *B;* the fixing point is at *A*. The small letters call attention to details of construction. *2*, the second piece of apparatus used by Aubert and Foerster. The flat strip rotates on the axis at *u* and the rider, *b*, bearing the two spots, is adjustable. *3*, the third piece of apparatus used by Aubert and Foerster and the original perimeter. The device shown in *2* was similar, but the arm was not curved.

of this first series of observations were as follows: "The further a figure is from the fixing point, just so much larger must it be to obtain recognition. The recognition for form does not decrease in concentric circles but declines more rapidly above and below and more gradually internally and externally." Realizing many of the objections to the method employed and the limitations inseparable therefrom, Aubert and Foerster set out to determine: "At what distance from the retinal center will two points of a certain size and definite distances apart, on the various meridians, be recognized as two distinct points?" They based their plan of using a two point test object on the experiments of E. H. Weber, who used two points to determine the fineness of sensation of the human integument, and on the plan of Hueck, who wished to know "how the sensitivity of the retina for images of objects decreases from the center toward the sides." Hueck fastened a test object to a pin at the center of an arc, turned his eye along the arc until the object disappeared and read the degree point at which he was looking at that moment.

Feeling that a curved arm could not be properly illuminated, Aubert and Foerster affixed a flat strip to the top of an upright in such a way that it could be rotated about the fixing point to try out the various meridians (fig. 4[2]). Along this flat strip, from the periphery toward the center, slid a carrier with two white squares which varied in size and separation. From this second series of experiments they drew the additional deductions that follow:

> The blind spot is not inserted between the normal retinal elements but is to be considered as an actual deficiency. The fineness of retinal sensation declines toward the periphery quite differently in different eyes and even in the two eyes of the same individual. There is no relation between this decline and the adjustment of the eye for near or far. The retinal sense of space is analogous to the space sense of the skin.

Aubert alone then proposed to answer the following questions:

> In what relation to the colored surface does the color sense decrease?
> How does this decrease go on along the various meridians?
> How do the various colors behave against a white and a black background?
> How do the various colors behave?

The apparatus employed was similar to that used in the preceding series except that the arm was a semicircle instead of a flat strip. This instrument (fig. 4[3]) was later to grow into the perimeter of Foerster introduced in 1869. The findings in this important series of experiments were as follows:

> A colored square on a white ground appears black to the observer when it has reached a certain lateral position. This change is gradual and not sudden. Beyond the line where the color appears dark is another line where it disappears, but a white spot would still be recognized.

A red, blue, green or yellow square on a black background appears as white beyond the limit described.

The larger a colored surface is, the further from the centrum will its specific color be recognized.

The limits for recognition of colors vary with the meridians.

Recognition of specific color at different points of the retina passes over into recognition of the test object without specific color in direct relation to the degree of contrast presented by the color against its background.

Taking an average of the limits obtained for each color against a black and against a white background, a kind of compensation is seen, and, using these figures as a basis, Aubert concluded that there was little difference in the capacity of the peripheral retina to distinguish one color from another.

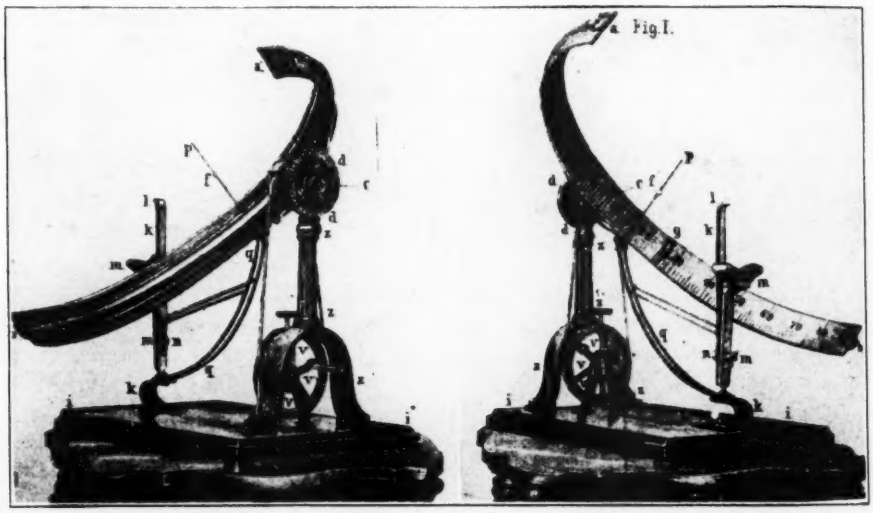

Fig. 5.—The Foerster perimeter of 1869. This is the logical development of the Aubert and Foerster device with the curved arm shown in figure 4[3]. The small letters indicate details of construction.

While the vexing questions concerned in testing with colored test objects have not been entirely settled even to this day, Aubert showed comprehensively that a black background favors red and blue test objects and constricts the field for green, while a white background constricts the field for red and blue and expands that for green. Yellow seemed to be uninfluenced by the change in background.

One must credit Aubert and Foerster with determining the limits of the field of vision for colors and white, with determining the small central area of "detail vision" and with showing the effect of the background on recognition of specific colors.

Foerster developed his well known perimeter from the last instrument and introduced it to the medical profession in 1869 (fig. 5). Until

this perimeter was used, examinations were limited mostly to the central 45° to each side of the fixing point. Efforts had been made to project the 60°, 70°, 80° and 90° circles on a flat surface by dropping perpendiculars from the respective degree marks of a semicircle and laying them off on a tangent flat surface. With the advent of the perimeter, more careful examination of the periphery came in, but what one knows now as campimetry went out because the perimeter was considered the perfect instrument, good for every purpose and every case. This idea has not to this day been dispelled and is responsible for much of the pessimism that inevitably follows efforts to examine blind spots and defects of the central areas with this instrument. Strange to say, much that was well known about the characteristic defects of the visual fields resulting from glaucoma simplex was forgotten and remained so until Bjerrum introduced his method of examining the fields with small white test objects in series at distances of either 1 or 2 meters.

After 1860, case reports including studies of the visual fields appeared in numbers, and only the notable indications of the general use of such studies can be cited. Müller of Oldenberg, Germany, in 1861 reported a case of "Visus Dimidiatus" and included a report of the pituitary enlargement found at autopsy. Seven cases of this disease had been reported up to this time, but only in one had the effects on vision been noted.

Meanwhile, de Wecker in Paris had improved the technic of the art, and the simple blackboard of von Graefe had, prior to the introduction of the perimeter, been marked off in concentric circles beginning with 10° and running out to the limit. As 90° could not be laid out on a flat surface, perpendiculars were dropped according to the rectilinear method instead of using tangent values. As a result of using this method, study of the first 40° was fairly accurate, but the 50° circle was the limit of even approximate accuracy. J. Hirschberg, in 1874, employed a 2 foot (0.6 meter) square blackboard but realized the limitation of flat surfaces to about 45° of the patient's visual field. He was thorough and skilful. In 1875, he discussed the chiasm and used the term "campimetry" for the first time. Details of the fields associated with various lesions about the chiasm, hemianopia, choroiditis, etc., are all included in case reports from his clinic, nor was the blind spot neglected.

Scherk devised a perimeter in 1872 (fig. 6) in the form of a half sphere, and to get the light to the quarter sphere in use at any time the unused half was swung back out of the way on a hinge at the zero point, not the fixation point. Up to the time of Carter's instrument, which came into use in England the next year, the fixing point was 15° to the right or to the left of the zero point, with the blind spot at the zero point. This was to harmonize the anatomic picture with

the record chart. Something is to be said for the idea, but for many years now all apparatus has been designed with the fixing point at zero. Carter described his instrument in the *Lancet* in 1872 and seems to be among the first to go into detailed studies of the blind spot. This was

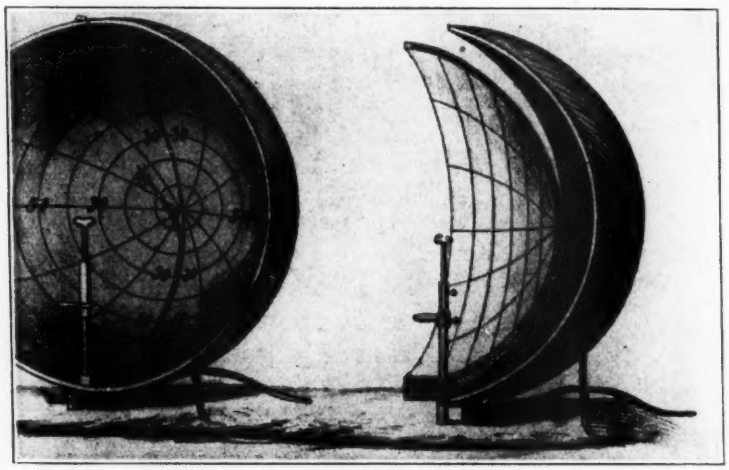

Fig. 6.—The Scherk perimeter of 1872. The half of the hemisphere not in use can be turned aside to provide proper illumination.

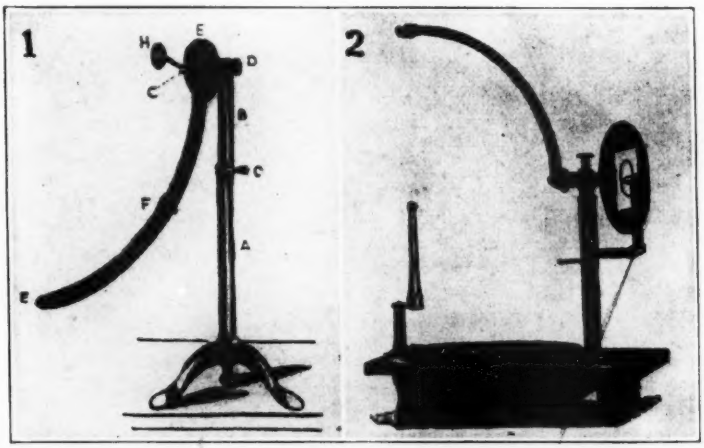

Fig. 7.—*1*, Carter's perimeter of 1872. The letters indicate details of construction. The fixing point is placed laterally with the blind spot at the axis of rotation, around which is a plate for outlining the blind spot. *2*, Smith's perimeter of 1881. On the stand lies a disk which can be attached and rotated, providing the first scotometer.

possible because he had a large zone at the zero area to serve as a background much like the present Ferree-Rand instrument (figs. 7^1 and 7^2).

Stilling brought out his ground glass globe in 1877. The patient looked into the globe through an aperture at one pole fixing the other pole while the test object, a rubber disk, moved from the 90° limit toward the center on the outer surface. The shadow cast by the rubber disk became the test object. The first registering device was invented by Stevens of New York (fig. 8), and was shown in 1881 at the meeting of the American Medical Association at Richmond, Va., and later in the same year at an international congress in London. Among those who saw this instrument was McHardy, a naval surgeon, who soon introduced his apparatus with catgut strings rolling around a drum as

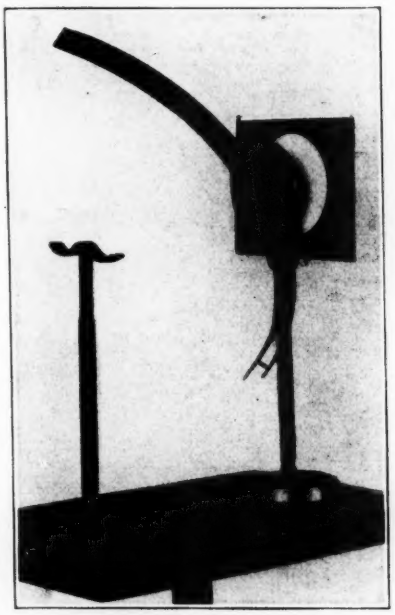

Fig. 8.—Stevens' registering perimeter of 1881.

the means of propelling the test object along the arm. These strings have been as troublesome to keep in order as the strings of the violin on a wet day or a grandfather's clock. He did illuminate his test object from behind by a gas jet with a reflector (fig. 9[1]). Priestley Smith brought out his simple but useful perimeter in 1883 and specifically described the principle later used in the scotometer: that of a test object revolving about the fixing point instead of moving from the periphery toward the center along the curved arm. In 1884, Albertotti (fig. 9[2]) showed his complicated device which could be operated by the patient, who might also record the point at which the test object was first recognized. He also used a fluorescent test object (barium sulfate) in dull light. In 1885, Dyer of Philadelphia introduced his perimeter on which a

spiral wire coil was rotated and the test object brought from the periphery to the centrum along any meridian very gradually and with constant speed. Meyerhausen introduced another complicated device in this same year. The illustration shows that the recording chart rotated with the arm, and that the point of recognition was stamped thereon by the examiner (fig. 9[3]).

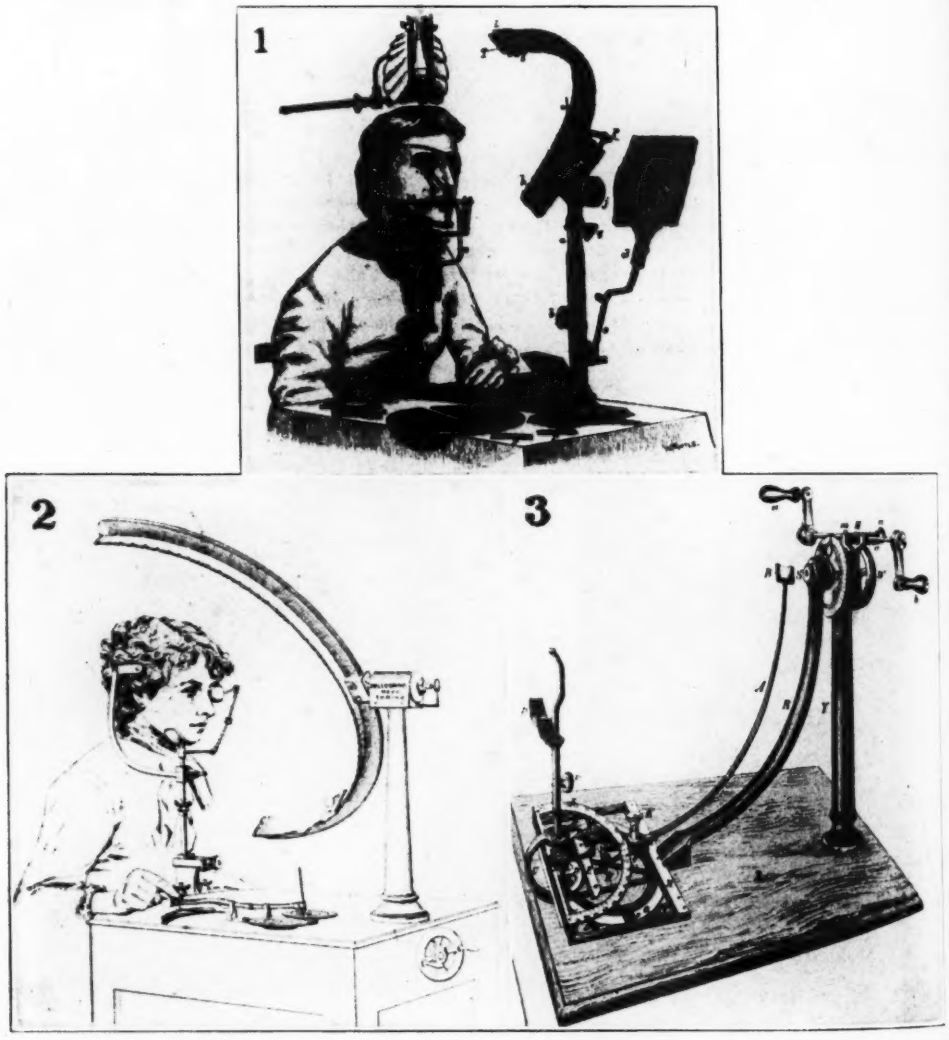

Fig. 9.—*1*, the McHardy instrument. The letters indicate details of construction. Note the crutch which is in contact with the face, beneath the eye, and the sliding attachment to clamp the teeth on. There is also a disk to be attached to provide a flat surface for examining the central areas. *2*, the elaborate device of Albertotti. In the patient's left hand is a crank handle similar to the one in the foreground. The patient turns the crank, and when the test object comes into view the point at which it appears is registered by the right hand. Other elaborations were included. *3*, the Meyerhausen perimeter of 1885.

The Schweigger hand perimeter came out in 1889, and in 1897 and 1898 practical but simple devices were described by Helmbold and Gagzow (fig. 10[1]). Giles of New York devised a simple and practical instrument in 1893, but few reproductions of it seem to have been turned out. Skeel planned his apparatus in 1900, and this was in use for many years. Zehender shut out the background, so plainly seen in the Helmbold apparatus, and placed all of the registering parts where the patient could not see them (fig. 10[2]).

One finds the first mention of work on the visual fields in America in an article published in 1874 by the pioneer of ophthalmology, Hermann

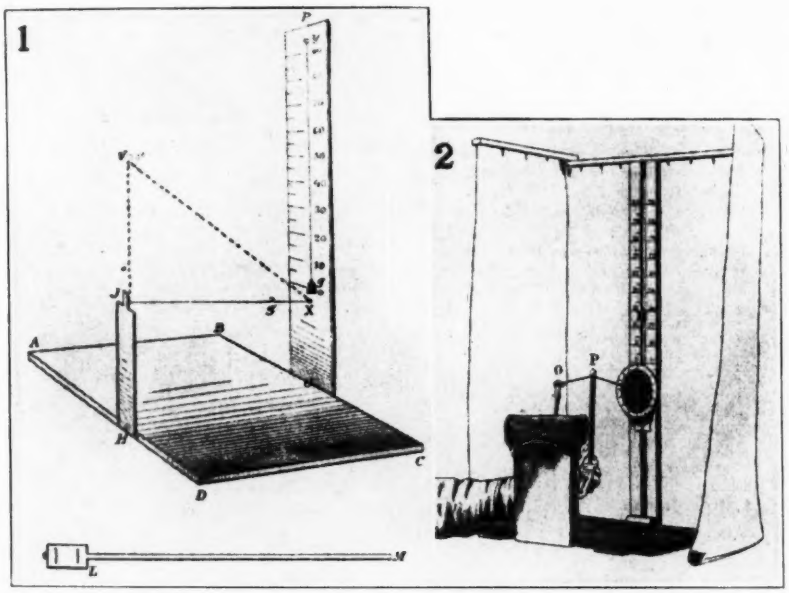

Fig. 10.—*1*, the very simple device of Helmbold. The letters indicate details of construction. Below is the test object carrier with a hook at the distal end. This is hooked into the loop marked *S*, and the degree points from the center are indicated by the position of the weight against the scale. The meridians can only be guessed at. This device was planned for the general practitioner's use. *2*, Zehender's improvement on the simple Helmhold apparatus. He shut out extraneous objects and added a disk at the center to give the different radii.

Knapp. He reported a case in which the fields were greatly contracted but made no mention of the means employed to determine the fact.

T. R. Pooley had a very fine article in the ARCHIVES OF OPHTHALMOLOGY in 1875, a report of a case of hemiopia, which is the first American mention of it. In 1879, Roosa discussed the visual fields as observed in cases of quinine poisoning and, in 1881, E. Gruening collected the data on all cases of quinine poisoning reported to that date

(eleven) and clarified the atmosphere of doubt as to what effects are due to quinine and what to the diseases for which it was given, often in huge doses.

The perimeter had attracted most clinicians, and from the date of its introduction until 1889 the central areas never received the careful study they deserved. In 1889, the work of Bjerrum, Meisling and Henning Ronne in Bjerrum's clinic in Copenhagen turned the thoughts of ophthalmologists to the midperiphery and came as near solving the problem of measuring the functions of the paracentral and peripheral retina as is probably possible. Oculists had acquired the habit of using a single test object on the perimeter or a series of white, red and green test objects, all of liberal size. This was comparable to using a single line of test type, say the 20/40 line, and feeling satisfied that visual acuity was normal if the patient could read this one line. Bjerrum and his associates had the benefit of a wider knowledge of the physiology of the eye than their predecessors and appreciated the "duplex" function of the central area and that of the periphery. He used a black velvet screen with appropriate but not conspicuous circles and radii, and by placing his patient at 1 or 2 meters and using small white test objects in series established a series of more or less concentric isopters which expressed the limits of the fields of vision for the various sizes of test objects. He could at the same time reduce his field limits to an area within the practical limits of a flat surface.

He did not confine his interest to the periphery but carefully investigated the central areas and disclosed the characteristics of the glaucomatous blind spot and central and paracentral areas. The trend of modern field work away from use of the perimeter toward careful examination of the central and paracentral areas against a flat surface began with Bjerrum.

In 1908, Bardsley introduced the scotometer, and though it was never used much, the idea of rotating the test object about the center was utilized in several simple campimeters, notably that of Smith, described in 1906. This consisted of nothing more than a rotating flat disk covered by black cloth. At the fixing joint was a white dot, and a wisp of wool of any color or size would adhere to the cloth surface of the disk and serve as a test object. It may be seen that all of these devices were clinical evolutions devised to facilitate examination and eliminate certain errors. Retinal physiology was not taken seriously into consideration.

In 1889, Bjerrum published his famous article which revolutionized perimetry and may be said to be the first application of the principles of physiologic optics. The mass rush from the perimeter to the campimeter which followed the publication of his article was much like the

earlier stampede from the campimeter to the perimeter. Many do not now use the perimeter, denying its value even in small degree. With appropriate test objects, the perimeter has a decided place in practically every examination of the visual fields. The preliminary survey of the fields can be made quickly with this instrument and valuable hints obtained as to the most productive method to be pursued to complete the details of the examination of the central and paracentral areas. Bjerrum placed examinations of the visual fields on a scientific basis, and every detail of his technic is sound and practical. As is always the case, there were many who went to unreasonable extremes in the size of the test objects used and in refinements of technic, but the present respect for examinations of the visual fields is largely due to Bjerrum and his co-workers. When one looks back on his reports one cannot but wonder at the long period elapsing before his method came into regular use. The British are indebted to Sinclair for early information concerning this technic and later to Traquair, who worked out practical details, putting the Bjerrum apparatus at the disposal of any one who would apply himself. In this country, Duane devised a practical screen with the Bjerrum diagram on one side and arrangements on the other for use in examinations of muscles. The first case report published in this country that I have found showing the Bjerrum defects of glaucoma simplex came out in 1909 and is the work of Dr. Harry Friedenwald.

Perimeters have always been made with the arm bearing the moving test object finished in dull black. Aubert showed the effect of light and dark backgrounds on colored test objects many years ago, but the first modern article on this important detail of color testing is that of Hess. He described the varying impressions made on the normal eye by a colored test object as it passes from the periphery toward the center. Thus red appeared first as a dark surface without color, then gray, next terra cotta, then yellow, then orange and finally red. It was a nice point to decide just when the recognition of red as a specific color began. To eliminate the recognition of a colored test object as a difference of shade rather than as a specific color, he recommended the use of a gray background of the same degree of lightness or darkness as the test object. This is the scientific basis for the present use of a medium gray coloring of the arm of the perimeter instead of the previous dull black. Holding the fixing eye steady is something of a problem in perimetry even with a normal eye, but with an amblyopic eye it is a real problem. When campimeters are used, the central areas are tested, and the accommodation should be relaxed by the proper glass to make the test as at infinity. The first effort to employ the steadying influence of the second eye while making sure that it did not take part in the examination was the binocular perimetry

of Schloesser. The eye to be examined was uncovered, but the other eye looked through a glass of a complementary color. The fixing point was a white disk and was observed by both eyes. If the test object was green, the second eye had a red glass before it which made the test object invisible. The error in this method was the inevitable color adaptation that occurred, but if each test period was brief good results could be obtained.

Soon after this, in Germany and later in this country, devices were introduced which permitted the second eye to fix but shut out a view of the test object. These were never practical. In 1904, Haitz introduced his charts, which are indispensable to modern clinical procedure. He used the simple Brewster stereoscope, which limited the usable area about the fixing point to 11° in each direction. At the fixing point of one of each pair of charts was a small colored dot about ½° in diameter. The dots were white, red, green, blue and in some editions, yellow. The technic was simple. If binocular vision was present, the eye to be tested was covered, but as soon as fixation with the uncovered eye was established the chart with the colored dot was momentarily uncovered. If binocular fixation was lacking, only the eye under examination was exposed, and the dot was covered by a test disk of small size, but, when fixed, this was lifted and immediately replaced. In this way, small defects could be found that otherwise were easily and habitually overlooked. The blind spot areas in glaucoma are so important that it seemed deplorable to allow such an excellent procedure to remain in disuse, and I urged use of the stereocampimeter in order that full advantage might be taken of the possibilities of this idea. Goldman has offered a plan whereby binocular fixation can be employed with the well known Duane screen or any of its modifications.

The literature of this branch of ophthalmology begins with the classic article by Thomas Young which appeared in the *Philosophical Transactions* of 1801. Purkinje's "Beobachtungen und Versuche zur Physiologie der Sinne" came out in 1825 and contains much important material on every phase of physiologic optics, which should be the basis of all examinations of the visual functions. Other articles of this type came from the hands of Griffin, Hueck, Volckmann, Hannover and von Kölicker, to say nothing of von Helmholtz' classic.

Arago's work on astronomy resurrected much of interest that had been published before 1800 and then forgotten or buried. The first article on the clinical uses of perimetry came from the pen of von Graefe in 1856, and it is a classic. Aubert and Foerster then began what one might call the first scientific investigations of extramacular areas. Many articles appeared from this time on, and case reports include statements of the condition of the visual fields. The Dutch oculists

contributed much to the study of the typical defects resulting from glaucoma simplex and von Graefe's classic article on Glaucoma (1869) includes a description of the defects of the visual fields in chronic cases that is amazing in completeness of detail even if one takes Bjerrum's article of 1889 as the standard.

The first book devoted exclusively to this line of work was put out by Wilhelm Schoen in 1874. The book is remarkable for the wealth of detail concerning the outlines of the visual fields in cases of various diseases of the eye, but the central and paracentral areas do not receive the attention they deserve. The wealth of detail given by von Graefe, Donders, Snellen and Haffmans concerning the defects associated with glaucoma simplex is forgotten, and only very meager details of the scotomas of retrobulbar neuritis are given.

The next book is by Pauli but, despite my best efforts, I have never been able to find it. It came out in 1875 with the title "Contribution to the Science of Visual Fields."

Ole Bull wrote a book entitled "Perimetrie" in 1895, and this is much further advanced than its predecessors. The clinical portion of the book is preceded by a portion entitled "Physiological Preliminaries." Examination of the central and paracentral areas with reduced illumination is recommended to bring out the defects characteristic of glaucoma simplex.

The next book was by Baas, put out by Enke of Stuttgart in 1896. This is an excellent work which utilizes retinal physiology to an even greater extent. Peter of Philadelphia put out his book entitled "Principles and Practice of Perimetry" in 1916, and a second edition in 1923.

My work entitled "Visual Field Studies" appeared in 1926, and Traquair's very fine "Introduction to Clinical Perimetry" is a year younger.

In volume 2 of the Norris and Oliver "System of Diseases of the Eye," published in 1900, was an article entitled "Perimetry" by Wilbrand. This was a condensed classic and had a tremendous influence in this country. Wilbrand's other articles have appeared in German and in the process of translation have acquired an anonymity robbing him of the credit to which he is entitled.

Perimetry in the dark room begins with the observation of Haffmans that in cases of glaucoma the oblique line separating the seeing from the nonseeing retina first appears on the nasal side, and that if this feature cannot be demonstrated in ordinary light it can be easily shown by reducing the illumination. Ole Bull in his book also emphasized this point. No mention was made by either of dark adaptation during this process. Albertotti used a fluorescent test object in the dark room. Perimetry of the dark-adapted eye was thoroughly studied

by Stargardt and others in a variety of conditions, among which was glaucoma simplex. This procedure is closely associated with the effect of various diseases of the eye on dark adaptation, which does not belong in this discussion.

From time to time, perimeters have been devised with electric illumination of white and colored mires of variable sizes, but none ever gained recognition because the conditions created were not comparable in any way to those under which all previous experience had been garnered.

Recently Passow urged again the examination of the blind spot and paracentral areas if glaucoma is suspected and claimed that this most discriminating test at one's disposal should be carried out in the dark room with the adapted eye. He has devised an apparatus which permits a spot of white or colored light of whatever size and light strength desired to be used as the test object.

From this review it is evident that all of the early methods employed in examinations of the visual fields were dictated by clinical experience. To avoid features found objectionable in one plan or apparatus, another was introduced, to give way in turn to another. It is quite unreasonable to expect any medical or scientific group to design deliberately an apparatus or a technic in advance that would measure up to all demands and conditions. It is somewhat surprising, however, to note how little attention was paid to retinal physiology, which should be the basis of every test and every piece of apparatus. Not until Bjerrum brought out his entirely practical and truly scientific method did the tide turn from the eminently practical to a combination of clinical experience with physiologic optics. In the very early days of ophthalmology one finds that the only test thought necessary to determine the condition of the retina was a set of test letters, because vision was considered a simple function that differed as one passed from the fovea to the periphery only in acuity, not in quality. By whom it was realized that something else than test letters was required, I cannot say, but only the paracentral areas were explored by means of the simple flat surface at first employed. When this had been worked out, the zones farther away from the center and finally the extreme periphery were included by the perimeter.

After Schultze discovered the rods and cones in the retina, an immense amount of work was done to explain the function of each end-organ. Retinal physiology, at first apparently so simple, had become most complex, and the apparatus designed for testing the added functions might be said to comprise a new department rather than an expansion of perimetry.

Recently, an effort has been made to determine, if possible, the very earliest stages of the breakdown in retinal functions which in comparatively late stages produces central and paracentral scotomas. To do this, the determination of the minimum of light necessary to make a retinal impression and the determination of the minutest light difference that can be observed have been added to the time-honored methods of perimetry and campimetry. The status of perimetry has also changed because of the introduction of other apparatus and a better understanding of disease conditions. The early ophthalmologists lacked many of the things one now enjoys and were compelled to depend on perimetry to differentiate glaucoma simplex from other conditions. Today, the ophthalmoscope usually excites one's suspicions, and the tonometer confirms or rejects them, but there is no real index of progress or loss except the results of a careful study of the blind spots and the finger-like defects extending from them around the macular area. Retrobulbar neuritis and the various hemianopias cannot be diagnosed except by studies of the visual fields because changes in the optic disk occur only late, or, as in lesions behind the thalamus, not at all. It is certain that fewer examinations of the visual fields are made now as a matter of routine, but with excellent apparatus and perfect illumination, those made are to the point and properly interpreted.

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News and Notes

SOCIETY NEWS

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom was held at The Royal Society of Medicine on April 2 to 4. A morning session was devoted to the discussion of "Ophthalmia Neonatorum." The following papers were among those presented:

"The Light Threshold in the Macular and Perimacular Regions, with Some Considerations upon the Standardization of the 'Light Threshold Test'," Mr. Ransom Pickard.

"Unilateral Melanosis of the Sclera and Uvea with Development of Sarcoma," Mr. Frank Juler and Mr. Frank W. Law.

"Detachment of the Retina with Vitreous Hemorrhage," Mr. A. Affleck Greeves.

"Ophthalmoscopic Estimation of the Calibre of Retinal Arteries in the Living Eye," Mr. Humphrey Neame.

"The Etiology and Treatment of Phlyctenular Keratitis," Mr. Arnold Sorsby, Dr. R. Hamburger and Dr. L. R. Behnam.

"Radium Necrosis of the Cornea," Mrs. Philippa Martin.

"Late Results of Corneal Grafts," Mr. B. W. Rycroft.

"Experimental Heterogenous Corneal Grafts," Mr. J. W. Tudor Thomas.

German Ophthalmological Society.—"The Importance of Heredity for Ophthalmology" will be the principal subject at the meeting of the German Ophthalmological Society to be held at Heidelberg on July 6 to 8. The following discussions will be presented: "Inheritance from a General Biological Standpoint," Professor Oehlkers, Freiburg; "Inheritance from a General Medical Standpoint," Professor von Verschuer, Frankfurt; "Inheritance in the Domain of Ophthalmology," Doz. Dr. Waardenburg, Arnhem; "The Responsibility of the Oculist in the Prevention of Inherited Diseases," Professor Clausen, Halle.

Philadelphia County Medical Society, Eye Section.—The following program was presented at the scientific meeting of the Eye Section of the Philadelphia County Medical Society on April 2:

"Retrobulbar Neuritis" (presentation of patients from Wills Hospital), Dr. R. S. Minton; "Chorioretinitis with Unusual Characteristics" (presentation of patients), Dr. J. M. Rosenthal; "Nodular Keratitis" (presentation of patient), Dr. Hunter W. Scarlett; "Two Cases of Hemorrhagic Retinitis Yielding to Removal of Focal Infection," Dr. David J. Boon; "Detection of Pituitary Tumor from Ophthalmic Findings," Dr. George H. Denny; "Retinal Angiostenosis in a Young Adult: Suspected Pituitary Tumor" (presentation of patient; discussion by Dr. N. W. Winkelman and Dr. Stuart N. Rowe), Dr. Sidney L. Olsho.

French Ophthalmological Society.—At the next meeting of the French Ophthalmological Society, the friends, colleagues and pupils of Victor Morax will place his portrait in the ophthalmic department of the Lariboisière Hospital, where Morax worked for more than twenty-one years. Subscriptions should be sent to Georges Masson, 120 Boulevard St. Germain, Paris. A reproduction of the portrait will be sent to all who subscribe 100 francs.

Société d'Ophthalmologie de Paris.—Dr. J. Gallois, 132 Avenue de Wagram, Paris, will appreciate receiving correspondence from ophthalmologists on the subject "Floating Bodies in the Vitreous," to be discussed at the annual meeting of the Société d'Ophthalmologie de Paris in November.

CORRECTION

In the article entitled "The Virus of Inclusion Conjunctivitis: Further Observations," by Dr. Phillips Thygeson and Dr. William F. Mengert, in the March issue (ARCH. OPHTH. **15**:377, 1936), the term "inclusion folliculitis" in the first line of the legend for figure 2, page 385, should read "inclusion conjunctivitis," and the legends for figures 6 (page 397) and 7 (page 402) are reversed.

Obituaries

HERMANN WILBRAND, M.D.
1851-1935

Hermann Wilbrand died in Hamburg, Germany, in September, at the age of 84 years. He was at first an assistant to Förster, under whom he learned to use the perimeter and received the incentive to study the course of the optic pathways in the brain. In 1879 Wilbrand became a resident of Hamburg, and the unusually rich material in nervous diseases at the St. Georg Hospital interested him in the neurology of the eye. During this period he was aided by anatomic investigations of the brains of patients with nervous diseases by his friend Henschen, of Uppsala. He then started to publish that monumental work "Die Neurologie des Auges," which appeared in seven volumes and which though carrying the names of Wilbrand and Saenger was practically the work of Wilbrand. Ophthalmologists in this country know Wilbrand best because he contributed the chapter on "Perimetry and Its Clinical Value" to Norris and Oliver's "System of Diseases of the Eye" in 1897. This chapter consisted of 140 pages and was translated into English by Dr. Thomas R. Pooley.

Wilbrand was an unusually modest man who rarely visited congresses. In recent years he suffered many illnesses but was able to continue his literary work. Through his death ophthalmology has lost an investigator of importance. His writings on the relationship of ophthalmology and neurology will be regarded as authoritative for many years to come.

ARNOLD KNAPP.

RICHARD DEUTSCHMANN, M.D.
1852–1935

Richard Deutschmann died in Hamburg, Germany, on Nov. 19, 1935, at the age of 83. He will best be remembered as the one who through many years kept up his interest in operations for detachment of the retina at a time when the surgical treatment of this condition had been practically given up by ophthalmologists. His interest and reputation attracted many patients with this unfortunate malady from all over the world.

Deutschmann received his early training in ophthalmology in Göttingen under Leber, where he did research work on sympathetic ophthalmia; he was one of the co-founders of the migratory theory, which for a time played such an important rôle in discussions of this baffling disease. At this time he also became interested in detachment of the retina, a subject in which later, as previously stated, he was to become so prominent. The various methods of treatment for detachment of the retina which he attempted consisted in the replacement of the vitreous of the patient with that of an animal, incision (relaxation) of the retina, chemical cauterization and subconjunctival treatment. Gilbert (*Klin. Monatsbl. f. Augenh.* **96:** 114, 1936) states that Deutschmann realized at an early date that closure of the hole was essential, and that he obtained favorable results in 25 per cent of his cases.

Deutschmann was also one of the first to become interested in nonspecific protein therapy. He helped in producing a serum composed of hormones from the sexual glands which was found of use in recurrence of cancer.

ARNOLD KNAPP.

W. ADAMS FROST, M.D.
1853-1935

W. Adams Frost died on Oct. 25, 1935, at the age of 82. He retired from practice thirty years ago. He became identified with St. George's Hospital in London in 1870 and later worked at the Central London Ophthalmic Hospital, where he succeeded McHardy. At St. George's Hospital he was assistant to Mr. R. Brudenell Carter. He was also on the staff of the Royal Westminster Ophthalmic Hospital for many years. Later in life he was attacked by glaucoma, and toward the end he became practically blind. His important contribution to ophthalmology was his magnificent "Atlas of the Fundus Oculi." In this he had the assistance of that unusual artist A. W. Head. This atlas has been the best on the market for many years, and even today the plates are unsurpassed.

Dr. Frost acted as librarian of the Ophthalmological Society of the United Kingdom for twenty-one years and made the Bowman Library one of the best ophthalmologic libraries in existence. He contributed many papers to the *Transactions of the Ophthalmological Society of the United Kingdom,* an exhaustive article on pulsating exophthalmos published in 1883 being of particular interest.

ARNOLD KNAPP.

A. S. PERCIVAL, M.D.
1863–1936

A. S. Percival, an English ophthalmologist, died on Jan. 9, 1936, at the age of 73. After graduating in medicine, he studied ophthalmology. He started practicing at Newcastle, where he remained for the rest of his life. During this entire period he was connected with the Newcastle Eye Hospital. He was one of those unusual ophthalmologists to whom a knowledge of mathematics came easily. One of his early articles on periscopic lenses appeared in the ARCHIVES in 1901, and another and final paper on this subject appeared in this journal in May 1934.

Percival had an original and active mind; he always deplored the ignorance of oculists concerning mathematics. In his publications he assumed that the readers had a knowledge of this subject, so that his books are not easily read. He wrote "Optics—A Manual for Students" in 1899, "The Prescribing of Spectacles" in 1910, "Geometrical Optics" in 1913 and "Practical Integration" and "Perspective." He was also interested in light sense and in miners' nystagmus. He invented a small apparatus for measuring light difference and light minimum.

In his early days in Newcastle Percival took an active part in the papers and discussions of the local medical societies. In 1893 he received the degree of Master of Arts from the University of Durham. He was interested in refraction to the end of his career, and particularly in cases associated with defects of muscle balance.

Percival founded a small society named "Sphalma," the purpose of which was for the members to derive benefit by a discussion of their mistakes and failures without risk of publicity. He was president of the Section of Ophthalmology of the British Medical Association at its annual meeting in Glasgow in 1922, where he opened the discussion on heterophoria.

ARNOLD KNAPP.

This obituary is based on one by Alexander MacRae in the British Medical Journal (**1**:89 [Jan. 11] 1936).

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE OCCURRENCE OF CILIARY PROCESSES ON THE IRIS. A. B. REESE, Am. J. Ophth. **18:** 6 (Jan.) 1935.

Reese finds that not infrequently one or all of the fully developed ciliary processes may arise from the posterior surface of the iris instead of from their usual sites on the zona ciliaris. They differ from those arising from the ciliary body by being covered with a double layer of pigmented epithelium at their base, at which point the dilator muscle is interrupted. There is usually a bridge between the iris and the processes of the ciliary body, beneath which may be a recess having the appearance of a cyst. These processes in the iris may cause a congenital irregularity of the pupil, or their excision may cause undue reaction and even result in sympathetic ophthalmia.

W. S. REESE.

Bacteriology and Serology

NONINFECTIOUS CONJUNCTIVAL ALLERGIES CONSIDERED IN REGARD TO GENERAL ALLERGY. KOUTSEFF, Bull. Soc. d'opht. de Paris, April 1935, p. 267.

Certain authorities believe that conjunctival allergy is due to a true relationship between the causal agent, the allergen, and the clinical symptoms (Wood); others feel otherwise (Kruckmann). Koutseff has attempted to answer this question by studying the allergic manifestations of the conjunctiva in relation to the entire organism. The methods used were two: he attempted to suppress the allergen, and he brought the allergen into direct contact with the eyelids or conjunctiva to produce a reaction. One patient with asthma who was sensitive to the castor oil plant had a marked local reaction on application of the protein directly to the eyelids. The conjunctival allergies are of two types: follicular, or eczematous, and edematous. The first group are found related to the cosmetics and pomades; the latter group, to air-borne allergens. In the former the Prausnitz-Küstner reaction is always negative, while in the latter group it is always positive.

L. L. MAYER.

Biochemistry

STUDIES IN THE PROTEIN CONTENT OF THE HUMAN AQUEOUS. E. SELINGER, Am. J. Ophth. **17:** 1130 (Dec.) 1934.

Selinger reviews the literature, discusses the diagnostic significance of the protein content of the aqueous, and gives the following conclusions:

"1. Certain therapeutic agents cause a dilatation of the blood vessels of the eye and an increase of the permeability of the vessel walls, thus

increasing the protein content of the aqueous. Immune bodies, which are found only in minute quantities in the normal aqueous, are found in large quantities in the plasmoid aqueous.

"2. It is possible to differentiate a papilledema from a papillitis by the protein content of the aqueous. The protein content of the aqueous is normal in choked disc, even though retinal hemorrhages and grayish areas of nerve-fiber degeneration are present. Papillitis of inflammatory and noninflammatory origin has a definitely increased protein content of the aqueous. This test should be of value as an additional diagnostic aid when one is in doubt as to whether one is dealing with a case of papillitis or papilledema.

"3. An increased protein content of the aqueous is definite evidence of an active inflammation, or other active pathological process, characterized by the transudation of a fluid high in protein constituents. This test can be used, in doubtful cases, to differentiate an active from a quiescent process.

"4. Transudates vary in protein content with their location. Those in the optic nerve are poor, while those in the retina and choroid are rich in protein constituents."

W. S. REESE.

FURTHER EXPERIMENTS ON ACTIVE PRINCIPLES IN THE OCULAR TISSUES. K. VELHAGEN, Arch. f. Augenh. **109**: 195, 1935.

Velhagen has reported the presence of active chemical substances in the eye in a number of previous papers. He has claimed that these substances are similar in their depressor action to histamine and acetylcholine. Recently Braun (*Arch f. Augenh.* **108**: 341, 1934) has criticized this work, and Velhagen's present article deals with further experiments designed to prove his original contentions.

He made use of a preparation of the back muscle of the eel in an experiment of the type described by Fühner. A piece of this muscle is sensitized by a previous administration of physostigmine. Following this it responds with contractions to a very dilute solution of acetylcholine when a small amount of physostigmine is added to the water bath in which the preparation is held. With such a test Velhagen was able to demonstrate the presence of acetylcholine in the retina and uveal tract.

F. H. ADLER.

Conjunctiva

MALNUTRITION IN RELATION TO ULCERATION OF THE CORNEA IN GONORRHEAL OPHTHALMIA NEONATORUM. C. E. WALKER JR., Am. J. Ophth. **17**: 1146 (Dec.) 1934.

Walker reviewed all cases of corneal ulcer in infants treated at the Massachusetts Eye and Ear Infirmary during the years from 1923 to 1933, inclusive, and a series of 100 consecutive cases (1927-1928) in which smears were positive for gonococci. Particular regard was paid

to health, weight, prematurity and gastro-intestinal disturbances. He states the following conclusions:

"1. Aside from the accepted local treatment, careful attention should be directed toward the dietary regulation of patients with ophthalmia neonatorum.

"2. Premature infants seem to be more susceptible to corneal ulcers than full-term babies.

"3. Gain in weight seems to be inversely proportioned to corneal involvement and progress in the infant."

W. S. REESE.

Cornea and Sclera

THE HISTOCHEMICAL REACTION OF THE DISEASED CORNEA. Y. SUGITA and S. SUGITA, Arch. f. Ophth. **134:** 175 (Aug.) 1935.

In a previous paper (*Arch. f. Ophth.* **130:** 488, 1933), Y. Sugita reported a histochemical difference between corneal epithelium and stroma: The reaction of the epithelium varied from neutral to slightly acid, while that of the stroma was alkaline. Now the authors report that in a state of inflammation the reaction of the stroma shifts toward the acid side. The shift was demonstrated by staining corneal sections with methylthionine chloride (methylene blue) and pyronine and also by the intracorneal injection of indicators in vivo.

P. C. KRONFELD.

KERATITIS NUMMULARIS DIMMER. J. SZEKELY, Arch. f. Ophth. **134:** 184 (Aug.) 1935.

About a year ago Salzmann gave a detailed and accurate description of keratitis nummularis (*Arch. f. Ophth.* **132:** 399, 1934). An assistant, Szekely, now reports what he calls the first case in which both eyes were affected at the same time. He has overlooked the report of a very similar case by W. Y. Chen in the *Chinese Medical Journal* (**48:** 890, 1934). He also describes a case of keratitis vesiculosa Vogt which developed into typical keratitis nummularis. Another possibility is the development of typical keratitis punctata superficialis from vesicular keratitis. From 1934 to 1935 twenty-five cases of keratitis nummularis were seen in the University Eye Clinic in Graz, Austria.

P. C. KRONFELD.

HISTOLOGIC OBSERVATIONS IN A CASE OF FAMILIAL DEGENERATION OF THE CORNEA. J. URBANEK, Klin. Monatsbl. f. Augenh. **94:** 382 (March) 1935.

A man aged 28 had presented familial degeneration of the corneas since his fourth year. His mother and grandmother suffered from the same affliction. His eyeballs were examined histologically after he succumbed to a fatal accident. It could be shown by divers methods of staining that the familial degeneration of the corneas consisted in deposition of fatty acids and their esters beneath the epithelium, where Bowman's membrane had been destroyed. The anterior elastic membrane was entirely absent in both eyes, which is contrary to the findings in E. Fuchs' case.

K. L. STOLL.

Experimental Pathology

A Review of the Subject of Artificially Produced Hypotony. F. Poos, Arch. f. Augenh. **109:** 162, 1935.

Poos reviews the facts relating to experimental production of hypotony, and points out that there is no specificity in the action of the various drugs or chemicals employed, but that one and all owe their effects to a toxic action on the walls of the intra-ocular capillaries. In his opinion, there is no difference in mode of action, so far as the production of hypotony is concerned, between a contusion, a subconjunctival injection of epinephrine or pilocarpine hydrochloride and an infusion of a hypertonic solution into the blood.

In chronic inflammatory conditions the long-standing hypotony is due, he believes, to a disturbance in the mechanism of fluid balance.

F. H. Adler.

General Diseases

The Chief Preventable Blinding Diseases of Childhood. R. E. Wright, J. Madras Univ. **6:** 172 (Aug.) 1934; **7:** 1 (Jan.) 1935.

LECTURE I. DEFICIENCY DISEASE AND TRACHOMA

Keratomalacia.—The first symptom of this disease is night blindness, and the earliest obvious sign is a peculiar smokiness of the conjunctiva. With progression of the disease, marasmus sets in. There are a diminution of secretion and an abnormal dryness of the nasal, oral and pharyngeal membranes. Usually the conjunctiva becomes dry, wrinkled and greasy-appearing, and the cornea becomes dull, lusterless and finally opaque, eventually undergoing necrosis and ulceration, with loss of the eye, if untreated. Occasionally one finds a wet, dull, red conjunctiva. Bitot's spots are present adjacent to the nasal and temporal limbus. The patients may be restored to health but remain blind because of the resulting dense leukomas of the cornea or because of staphylomas.

Even in extreme cases the child can be restored to comparative health by cod liver oil. If the child cannot take the oil by mouth, it is possible to save its life by wrapping it in a flannel binder soaked in the oil. The disease is apparently due to a deficiency of vitamin A, although vitamins B, C and D may also be concerned. No remedy as effective as crude cod liver oil has yet been found, and the average quantity necessary for small children need not exceed 7½ minims (0.46 cc.) per day, by mouth.

The incidence of cases of total and practical blindness due to keratomalacia is approximately 3 per thousand cases of ocular disease seen at the hospital.

Prevention of this disease, which is probably responsible for more blindness in young children in India than any other single condition, depends entirely on an improvement in diet and in living conditions.

Trachoma.—Wright believes that the early stages of this disease are so indefinite that one can only record a suspicious case as that of a trachoma-like condition and should not subject the patient to some of the appalling treatments at present in vogue, which produce changes in nontrachomatous tissue that may be mistaken for later stages of the disease itself.

In India, trachoma is a disease more frequently found in Mohammedans (especially the women) than in Hindus (in whom it is more common among the Brahmins). Its incidence varies from 3.5 to 50 or 75 per cent of the total number of patients with ocular disease treated in several widely scattered ophthalmic teaching centers in India. It is associated with overcrowding, indoor living, confined dark unventilated housing and the purdah rather than with poverty per se. It appears to be more common in groups of Indians who habitually drape the head and face with clothing, and it is not particularly prevalent among rural Indians who go practically naked. It seems to be a disease of the "great unwashed." While not important as a cause of blindness in children of preschool age, yet it is one of the factors responsible for preventable blindness in young people. Treatment is ineffective unless patients are followed up, for relapses are common. Copper sulfate is still one of the most generally used and effective medicaments.

The prevention and eradication of trachoma in India depend on better housing conditions, social education and the emancipation of women. This is, of course, a public health problem.

LECTURE II. VENEREAL DISEASE AND CORNEAL TRAUMAS AND ULCERATIONS

Ophthalmia Neonatorum.—While this disease accounts for about 10 per cent of the total blindness in England and Wales, it is not the greatest cause of preventable blindness in Indian children, its incidence averaging about 2.1 per thousand cases of ocular disease in hospitals. Even when it occurs it seldom produces blindness. In India, one probably deals with a less virulent strain of the gonococcus than that responsible for the disease in England.

Credé's method of prophylactic treatment, if properly carried out, is still the most certain way of preventing the disease.

Congenital Syphilis.—Parenchymatous keratitis (interstitial keratitis or uveitis anterior) is one of the most important of the stigmas of congenital syphilis producing blindness. Its incidence averages 1.5 per thousand cases of ocular disease in hospitals. Although not common during the first five years of life, it may be regarded as a disease of childhood since the infection producing it is present at birth. Its prevention depends on (1) the prevention and cure of syphilis in adults since it is very amenable to treatment and (2) a greater emphasis by the government on the control and cure of this disease rather than of tuberculosis and leprosy, which tend to disappear naturally with the betterment of social conditions.

Smallpox.—Of 726 opacities of the cornea (leukomas and nebulae) seen during 1929, 59 followed smallpox, 2 followed ophthalmia neonatorum, 92 followed the use of irritant remedies, 68 followed injuries, 54 followed ulcers, 41 followed trachoma, 18 followed keratomalacia, 14 followed syphilis, and 378 were of unknown origin. Of 143 anterior staphylomas (ectatic corneal scars), 16 followed smallpox, 35 followed the application of irritant remedies, 31 followed injuries, 2 followed ulcers, 6 followed keratomalacia, and 53 were of unknown origin.

As, in the six years from 1928 to 1933, from 37 to 47 per cent of the children surviving at 1 year escaped vaccination in the Madras

Presidency, it is apparent that, with more universal vaccination, the cases of blindness due to smallpox could be prevented.

Irritant Remedies.—Approximately 6 per thousand persons of the hospital population were rendered partially or totally blind by irritant remedies in 1929. About one third of these were totally blind. Blindness from this cause, peculiar to children in the East, is due to superstition and ignorance. The various irritants used include tobacco juice, red-hot coals, alum, chillies, urine and dung.

The only method of preventing such practices is that of education by means of posters, lectures, leaflets and other types of health propaganda.

LECTURE III. HEREDITARY DISEASES

The cases of hereditary disease causing impaired vision or blindness number 4.55 per thousand ophthalmic patients in hospitals. The cases may be grouped (Treacher Collins) into (*a*) those in which the tissue involved has from the first been imperfectly developed (buphthalmos, ectopia lentis, colobomas, microphthalmos, anophthalmos, congenital cataract, tower skull, etc.) and (*b*) those in which, after full development, degeneration sets in (the tapetoretinal degenerations, progressive macular degeneration with or without dementia and with or without pigmentation, familial choroiditis, Tay-Sachs disease, gyrate or total atrophy of the retina and choroid, hereditary optic atrophy, familial corneal degenerations, etc.). Gowers devised the term "abiotrophy" to characterize the latter group.

In India, retinitis pigmentosa seems to be definitely associated with consanguineous marriages.

Prevention is possible only by sterilization of those known to be transmitters of hereditary blinding diseases. In the course of time this would accomplish much good.

W. F. DUGGAN.

THE LIGHT SENSE IN ARTERIOSCLEROSIS AND MALARIA. G. BORSELLINO, Arch. di ottal. **42:** 199 (July-Aug.) 1935.

Borsellino reviews the literature on examination of the light sense in general diseases.

In a series of examinations of supposedly healthy patients, one instance was found of marked reduction of light sense, and further examination showed the patient to be a subject of diabetes. In a series of eleven patients with arteriosclerosis between the ages of 50 and 73, investigations of the light sense were made with Nagel's adaptometer, after adaptation to the dark for thirty minutes. The light sense of each eye and that of both eyes together were recorded. The minimum recorded for normal persons by Accardi and Galante in the same clinic was 14,900 units. In the present series there was one patient whose light sense was above this minimum. The others all showed varying degrees of reduction. In one patient the light sense of one eye was as low as 2,000, while the average for the group (binocular) was 9,462. Patients who had definite fundus changes aside from arteriosclerosis were excluded, and most of the subjects had nearly normal vision. Some persons with a slight reduction in central vision showed a corresponding reduction in light sense, but others with normal central vision

showed an equal or even greater reduction in light sense. There was no relation between the reduction in light sense and the blood pressure, which varied considerably in the group. Borsellino believes that reduction in the light sense corresponds to diminution in the size of the retinal vessels with resulting disturbances of nutrition at the periphery of the retina and with possible accumulation of toxins in the retina.

In a second series of thirteen persons suffering from malaria a similar investigation was carried out. This time the Birch-Hirschfeld adaptometer was used with Borsellino's light sense as a control. In four cases the light sense was normal, and in the others definite reduction was present varying from one half to one ninth of the normal. Borsellino believes that the anemia present in these cases was largely responsible, although possibly an occlusion of some of the smaller vessels by the malarial parasites played a part.

S. R. GIFFORD.

Glaucoma

SHADOWS OF CALCAREOUS DEPOSITS SEEN IN ROENTGENOGRAMS OF THE AREA OF THE CHIASM IN CASES COMPLICATED WITH GLAUCOMA. K. VELHAGEN JR., Klin. Monatsbl. f. Augenh. **94:** 586 (May) 1935.

Thiel showed that certain atrophic processes in the optic nerve and certain degenerative diseases in the eye may be explained by shadows of calcareous deposits in the area of the sella turcica. The shadows may be observed on oblique exposures; they are caused by calcification of the walls of the carotid and the arterial circle of Willis or by lime deposits in the dura mater. The cases are usually marked by so-called pseudoglaucoma, which presents the appearance and functional disorder of simple glaucoma whereas increased tension can be neither found nor elicited. Velhagen reports seven cases observed at the University Eye Clinic of Halle, Germany. Three of the patients presented the appearance of "pseudoglaucoma"; all of them had hypertension as a consequence of a general physical disease, but the intra-ocular tension remained constantly below the normal standard. The shadows on the roentgenogram showed decided sclerosis in the sources of the ophthalmic artery. Similar conditions were found in the roentgenogram in two of fifteen cases in which genuine simple glaucoma was present which he examined for comparison. In two other cases there appeared a bridge in the sella in connection with the calcareous shadow and a unilateral bridge in the sella, respectively; in the first of these two cases, that of a woman aged 63, there was also the shadow of a sclerosed vessel in the sella.

The condition described may explain, in Velhagen's opinion, some of those cases of glaucoma in which the excavation of the optic nerve progressed although the intra-ocular tension had been lowered by an operation.

In these cases with an arteriosclerotic etiology, the removal of the roof of the orbit after Schloffer's method to free the optic nerve from surrounding pressure may be considered rather than decompressing ocular operations.

K. L. STOLL.

Lacrimal Apparatus

TREATMENT OF EPIPHORA WITH A STRONG SOLUTION OF SILVER NITRATE. G. S. VENGRZHENOVSKY, Sovet. vestnik oftal. **6:** 850 1935.

Vengrzhenovsky believes that the various anatomic and mechanical obstructions in the nasolacrimal duct (which he demonstrates with a number of roentgenograms of the latter) make treatment of the duct with liquid more effective than that by the old methods, for example, probing. He tried various strengths of silver nitrate and found a 10 per cent solution the most adequate. Three tenths of 1 cc. of a 10 per cent solution is instilled into the duct through the previously dilated lower punctum. The duct is then immediately syringed with physiologic solution of sodium chloride to neutralize the excess of silver nitrate. Of thirty-eight patients treated, thirty-one were cured. In the remaining seven the instillation was not done properly owing to fault of the patients. All the patients were examined rhinoscopically and for the patency of the duct after West. From one to four instillations of the solution at intervals of from five to eight days were sufficient. Only in four patients were pain and slight swelling of the region of the lacrimal sac observed, for one or two days.

Vengrzhenovsky recommends this method for use in cases of epiphora in which probing is not effective. The action of the silver nitrate he considers to be threefold, consisting in: (1) a narrowing of the lumen of the blood vessels; (2) a diminution of secretion and transudation and (3) limitation of the emigration of leukocytes.

O. SITCHEVSKA.

Lens

DINITROPHENOL CATARACT. D. G. COGAN and F. C. COGAN, J. A. M. A. **105:** 793 (Sept. 7) 1935.

The first case occurred in a man, aged 38; the second, in a married woman, aged 25.

The author's summary will suffice:

"In the two cases reported only therapeutic doses [of dinitrophenol] has been taken, and cataracts developed subsequently. Toxic neuritis also developed in case 1, but no other evidence of intoxication appeared in case 2.

"These two instances of rapidly developing cataract of a characteristic morphology occurred in relatively young persons in whom thorough study failed to reveal any etiologic factor other than dinitrophenol. The type of cataract which the first patient showed was, per se, suggestive of some systemic intoxication. The initial opacities occupied predominantly the posterior cortex but were not clearly demarcated from the surrounding areas. They progressed with extreme rapidity and formed a characteristic polychromatic luster in the zone of specular reflection."

W. ZENTMAYER.

CATARACTS FOLLOWING DINITROPHENOL. P. W. KNISKERN, J. A. M. A. **105:** 794 (Sept. 7) 1935.

The patient was a married woman, aged 37. She had had three children, one of whom had congenital cataract. The drug was used for about ten months when it was discontinued because the patient was pregnant. Incipient cataract was diagnosed by an ophthalmologist about fourteen months after the beginning of the treatment. Four months later the cataracts were mature.

Two factors other than the use of dinitrophenol are to be considered in this case: The cataracts developed late in pregnancy, and they occurred in a woman who had given birth to a child with congenital cataract.

W. ZENTMAYER.

CATARACT FOLLOWING THE USE OF DINITROPHENOL. N. K. LAZAR, J. A. M. A. **105:** 794 (Sept. 7) 1935.

Lazar's patient was a married woman, aged 44. She had been taking dinitrophenol for twelve years. The history indicated that there was evidence of cataract ten months previous to examination. The lenses showed many subcapsular striate opacities anteriorly with numerous small round white opacities deep in the central cortex. The posterior cortex resembled a postcortical saucer-shaped cataract.

A second case seen by Gifford is recorded. The patient was a married woman of 42. She had been taking dinitrophenol for several months. Three months after its use had been stopped, lenticular opacities began to develop, and about one year later there was intumescent cortical cataract in both eyes. One eye was successfully operated on.

Lazar makes the following comment: "A case is reported of bilateral anterior and posterior cortical cataract in a comparatively young person, coming on suddenly after prolonged treatment with dinitrophenol, with a great loss of vision over a period of less than four months, and showing a marked increase in the hyperopia. The well known fact that the drug causes a tremendous dehydration of tissue may hasten the maturity of lens opacities already present. An important fact is that, as shown by these and other reported cases, cataract may develop on average dosage of the drug, and without any general symptoms suggesting harmful effects. In the second case the opacities were first noted some time after the drug had been discontinued."

W. ZENTMAYER.

LATE DEVELOPMENT OF CATARACT FOLLOWING THE USE OF DINITROPHENOL ABOUT A YEAR BEFORE. T. D. ALLEN and V. M. BENSON, J. A. M. A. **105:** 795 (Sept. 7) 1935.

The patient was an unmarried woman, aged 25. The authors' summary gives sufficient details: "There was a history of the ingestion of approximately 73 Gm. of dinitrophenol over a period of about five and a half months, with the first evidence of cataract seen nine months after the drug was discontinued. The cataracts developed simultaneously, but one more rapidly than the other, one becoming complete within five months."

Both eyes were successfully operated on by the method of linear extraction.

W. ZENTMAYER.

THE IRIDOCORNEAL ANGLE IN OPERATIONS FOR CATARACT. R. ROSSANO, Bull. Soc. d'opht. de Paris, April 1935, p. 187.

A study of the angle was made in order to determine whether any reason could be found for the ascension of the pupil which frequently follows operations for cataract. The Troncoso gonioscope was used to evaluate changes in the arrangement of tissues in the angle. Twenty-three observations are outlined; the type of operation, the complications, if any, and the results are given in each case. In fourteen of these cases ascension of the iris was noted, and in six the rise of the iris was pronounced. In each of these six instances there was definite rupture of the hyaloid membrane, and in three of these, evidence of endothelium in the iridocorneal angle. Traces of iris pigment were found when ascension of the iris was present and not when the iris remained in its proper position.

L. L. MAYER.

HEREDITARY CATARACT, ESPECIALLY CATARACTA CENTRALIS PULVURULENTA. A. RUSSO, Ann. di ottal. e clin. ocul. **63**: 105 (Feb.) 1935.

Russo reviews previously reported cases of this type of cataract, which was first described by Vogt in 1921, and recalls the various theories concerning the pathogenesis of hereditary cataract and zonular cataract. He reports five cases of cataracta centralis pulvurulenta (embryonal nuclear cataract) in one family, the cases being studied especially with reference to possible etiologic factors.

Three of the patients were the only sons of the first patient through marriage to a first cousin. The fifth patient was the only surviving brother of the first patient. The five children of this brother were examined, but none showed lenticular changes. The lens opacities in the first patient and his brother consisted in a sharply circumscribed mass of shiny points surrounding the embryonal nucleus and veiling the embryonal sutures. In the children similar central opacities were present but, in a smaller central area, the white points were fused together in a peculiar formation resembling the crown (*chioma*) of a tree. In all the cases the opacities were bilateral and similar in the two eyes. The peripheries of the lenses were clear, and the eyes showed no other abnormalities except myopia in one case and hyperopia or hyperopic astigmatism in the others. Vision was reduced to from 2/10 to 9/10. Physical examination was in general without results. The blood calcium was 8.8 mg. in one case, but in the others was normal or nearly so. Two patients showed signs of rickets, which in one was marked.

The factor of consanguinity in the inheritance of this condition is well illustrated by the normal children of the first patient's brother, whose marriage was not consanguineous. The cataract was inherited as a mendelian recessive which remained latent in this family while becoming manifest in the issue of the two cousins. Since the opacity in these cases is stationary, Russo questions whether it should be considered as cataract and suggests the name "central pulvurulent opacity."

S. R. GIFFORD.

CLINICAL AND HISTOLOGIC STUDIES ON PLANAR CAPSULAR CATARACTS: REPORT OF CASES. M. BÜCKLERS, Klin. Monatsbl. f. Augenh. **94:** 289 (March) 1935.

Bücklers renders a short synopsis of the literature on capsular cataract, mentioning Otto Becker as the first to publish an exhaustive study on this topic. He divides the capsular cataracts into two groups: (1) primary capsular cataracts, to which belong (*a*) the anterior polar cataract with its transition into the pyramidal cataract and (*b*) the multiple capsular dots in juvenile and presenile cataracts; (2) secondary capsular cataracts, including (*a*) the capsular cataracts after contusions and perforations and (*b*) the planar cataract in the complicated and hypermature cataract. He reports three of his own cases and discusses them in detail, with these conclusions:

1. The capsule is closely attached to the capsular cataract; it shows no changes; the folds in the capsule develop by adaptation to the reduced volume of the subcapsular tissues, whereas the cross-section of the substance of the capsular cataract is thinner than that of the adjoining cortex.

2. The capsular cataract develops by proliferation of the capsular epithelium, which, from an unknown cause, forms spindle-shaped cells. Fine fibers are found around and between the spindle cells. These fibers stain like collagenous fibrils. A stratum of the same refraction index as that of the fibers was noted, which increased in volume as the number of cells decreased. This intermediary stratum was transformed into strands of a firm glassy mass in which finally only a few nuclei remained, which barely accepted the staining.

3. Amorphous flakes are present in the marginal areas of more recent capsular cataracts; they are supposed to be degenerative products of the epithelium.

4. The capsular cataract is a pseudometaplasia, although it stains like collagenous connective tissue.

5. A capsular cataract may be deemed an incomplete regeneration, and marginal indifferent zones may be looked for in the proliferating areas, which are the indifferent zones out of which a differentiation of the capsular epithelium originates. The term "subcapsular cataract" would be more correct than "capsular cataract."

K. L. STOLL.

Neurology

DISTURBANCE OF FUSION AFTER CONCUSSION OF THE BRAIN: REPORT OF CASES. P. A. JAENSCH, Klin. Monatsbl. f. Augenh. **94:** 470 (April) 1935.

Jaensch reports five cases of disturbed fusion of the type described by Bielschowsky as "group I." In these cases the breadth of fusion was abnormally small or fusion was absent, with resulting fatigue and occasional double vision. In each case the disturbance followed concussion of the brain. No conclusions were gained regarding the location of the fusion center in the brain or the course of its tract. The visual fields for white and colors were normal in three patients; in two

others the fields showed spiral-shaped concentrations. A lesion in the area of the calcarine fissure may be excluded; the lesion occurred in the descending portion between the optical cortex and the nuclei of the ocular muscles. Every patient with uncertain complaints of scintillation and double vision should have the breadth of fusion examined with Bielschowsky's universal prism apparatus or with Herschel-Landolt's double prism.

Jaensch gathers from his observations that disturbance of fusion after concussion of the brain is not rare, and that fusion must be examined before the patient's claims can be refuted. Genuine concussion of the brain may cause diminution or absence of fusion, contrary to Uhthoff's opinion; it may also lead to manifestation of anomalies of the state of rest and to double vision in which every sign of paralysis is absent. The earning capacity must be considered impaired 25 per cent when double vision cannot be corrected with prisms, because patients suffering from disturbances of fusion are obliged to wear a frosted lens.

K. L. STOLL.

AN OCULAR SYNDROME AFTER CONCUSSION OF THE BRAIN: REPORT OF CASES. J. KLAR, Klin. Monatsbl. f. Augenh. **94:** 500 (April) 1935.

Klar describes a type of patient who, after recovery from the acute symptoms of concussion of the brain, complains of persistent headaches. No objective neurologic symptoms can be found, not even increased pressure of the cerebrospinal fluid. This syndrome is found more often than neurologic conditions in men who have suffered injury to the skull. Twenty cases are reported and tabulated, with the following symptoms predominant: constant and persistent headaches, vertigo, scintillation, and obstruction of the visual field. A pathologic increase in the retinal arteries was demonstrated with the ophthalmodynamometer and a peripheral contraction of the field of vision with the campimeter. The hypertension in the retinal and cerebral arteries is considered to be centrogenous and caused by traumatic irritation of the vasomotor center located in the medulla oblongata. The contraction of the field of vision is of retinal origin and results from deficiency of oxygen or ozone (O_2), a consequence of the disturbance of the circulation by hypertension.

K. L. STOLL.

ANATOMIC CONTRIBUTION TO THE QUESTION OF RECURRING PARESIS OF THE OCULOMOTOR NERVE: REPORT OF A CASE. W. HANDMANN, Klin. Monatsbl. f. Augenh. **94:** 606 (May) 1935.

Handmann reports clinical and postmortem observations on a man, aged 52, with paralysis. Recurring paresis of the levator of the left eyelid was observed which finally became total and permanent. He attributes this disorder to a lesion in the oval portion of the left lateral large cell nucleus and to infiltration of the blood vessel in this area. Repeated vasomotor changes may have produced the recurrence of the paresis originally.

K. L. STOLL.

Operations

AUTOKERATOPLASTY OF MORAX. R. FRIEDE, Arch. f. Augenh. **109:** 243, 1935.

A case is reported of a young girl who had a round leukoma about 3 mm. in diameter in the center of the left cornea. Vision was reduced to perception of hand movements. The method of Morax was used in trephining the leukoma and a clear area of the cornea and transposing the two disks. In Morax' method the disks cut by the trephine are not complete sections but include merely the anterior two thirds of the cornea.

Friede made through and through disks and protected the lens and iris with small sheets of celluloid (*Matrizen*), which were introduced into the anterior chamber through a keratome incision before trephining. The disks stayed in place, and visual acuity was improved to 1/60.

F. H. ADLER.

Orbit, Eyeball and Accessory Sinuses

BENIGN OCULAR MELANOSIS. E. FILIPPI-GABARDI, Arch. di ottal. **42:** 225 (July-Aug.) 1935.

Filippi-Gabardi investigated three cases of benign melanosis, studying the condition with the slit lamp and in one case removing portions of the episcleral tissue for histologic examination. In the first case he observed a diffuse lead-gray coloring of the whole anterior segment. The iris was intensely pigmented, the pigment obscuring the normal markings. A halo of dense pigmentation was present about the nerve head. Seen with the slit lamp the pigmentation was most intense at the sites of the penetrating ciliary vessels. When the episcleral tissue was removed for biopsy, the sclera itself showed no gross evidence of pigmentation. The episcleral tissue on section showed granules of yellow-brown pigment, some contained in endothelial cells. There was a history of development of or at least increase of the peculiar pigmentation during early adult life. The second case was similar to the first except that the fundus was not involved. In the third case the pigment was localized to the upper half of the globe, and corresponding to this was an area of hyperpigmentation of the iris. In all three cases only one eye was involved. Vision was normal. Careful examination including that of the blood revealed no abnormalities. The author believes that the abnormal pigment originated in the uveal tract and reached the episcleral tissues through the penetrating vessels, which he thinks is proved by the fact that the sclera itself, except in the neighborhood of these vessels, was not pigmented. For practical reasons it was impossible to remove tissue from the sclera itself for biopsy. He believes that abnormal pigmentation of the uveal tract is congenital while that of the other membranes is acquired secondarily.

S. R. GIFFORD.

BLOOD PRESSURE IN THE RETINAL VESSELS IN A CASE OF PULSATING EXOPHTHALMOS. M. BAURMANN, Arch. f. Ophth. **134:** 192 (Aug.) 1935.

A case of typical traumatic pulsating exophthalmos (left eye) was under Baurmann's observation for fifteen months. During this period

the exophthalmos appeared, was treated by periodic compression of the carotid artery and finally disappeared. In this case Baurmann was able to study the mechanics of retinal circulation by frequent determinations of the retinal diastolic arterial and venous pressure with his dynamometer.

During stage I all the classic symptoms of pulsating exophthalmos were present. The venous pressure was increased in the involved left eye because of the direct communication between the carotid artery and the cavernous sinus. During the compression of the left carotid artery (and unavoidably also of the left jugular vein) the diastolic arterial pressure dropped to 56 while the venous pressure rose to 61. That the venous pressure actually became higher than the arterial was clearly brought out by the fact that the blood flow in the left retina became reversed. Immediately after starting the compression of the left carotid

	Right Eye		Left Eye		
Time of Observation	Diastolic Arterial Pressure	Venous Pressure	Diastolic Arterial Pressure	Venous Pressure	Comment
At the beginning of the treatment period (stage I)	62	21	62	49	
During compression of the left carotid artery	..	..	56	61	Blood flow in retinal vessels reversed
Six weeks later (stage II)	..	..	58	24	Left eye receding
During compression of the left carotid artery	..	..	52	24	Blood flow not reversible and more
During compression of the right carotid artery	..	..	30	..	
Two months later (stage III)	50	20	50	20	
During compression of the left carotid artery	46	20	46	20	
During compression of the right carotid artery	30	20	36	..	Patient collapsed several times during test
Six months later (stage IV)	66	20	66	20	
During compression of the left carotid artery	77	20	60	20	
During compression of the right carotid artery	60	20	64	20	

artery arterial blood entered the eye through the retinal veins, spread pulsatingly to the periphery of the fundus and was first seen as venous blood in the smallest retinal arteries, through which it left the eye. The same result was reproduced again and again. As soon as the compression ceased arterial blood began to enter through the arteries. This phenomenon Baurmann explains as follows: The compression raises the pressure in the left jugular vein to the level prevailing in the circle of Willis. This would mean no difference in pressure between the central artery and the central vein and therefore no retinal circulation. The ophthalmic artery has several anastomoses with the meningeal middle artery, in which the pressure must get very low during the compression of the left carotid artery. Thus it is possible that the pressure in the central artery becomes lower than that in the cavernous sinus and that the artery becomes the outlet for venous blood from the eye.

During the first few days of stage II the exophthalmos and the bruit disappeared. The venous pressure in the left eye returned to normal. The blood flow could no longer be reversed. Compression of the right carotid artery made the arterial pressure in the left eye sink to the level

of the venous pressure. The left eye, in other words, depended for its blood supply chiefly on the right carotid artery. Together with the disappearance of the pulsating exophthalmos the lumen of the left carotid must have become very much narrowed. During stage III this behavior became even more pronounced. The patient fainted repeatedly during the compression of the right carotid artery. Finally (stage IV) the pressure findings before and during compression returned to normal. "The left carotid artery became patent again." Baurmann recommends his method of determining retinal blood pressure before and during compression of the opposite carotid artery as a means to find out to what extent one carotid artery can act as a substitute for the other.

P. C. KRONFELD.

Pharmacology

LATER INVESTIGATIONS ON THE INFLUENCE OF TESTICULAR EXTRACT ON THE PENETRATION OF DRUGS INTO THE EYE. G. ROSSI, Arch. di ottal. **42**: 241 (July-Aug.) 1935.

The literature on absorption of drugs into the eye is reviewed, as well as the author's previous research, which gave somewhat conflicting results. In the present experiment rabbits' eyes were bathed in testicular extract for ten minutes, and then in solutions of sodium salicylate and potassium sulfocyanate for thirty minutes. Isotonic, hypotonic and hypertonic solutions were employed. Five minutes after this treatment the aqueous was aspirated and examined for its content of the drugs employed. Neither the tissue extract nor the solutions produced any ocular reaction except a slight hyperemia. The other eye was used as a control, the drugs being employed without the testicular extract. It was found that hypotonic solutions of sodium salicylate penetrated into the aqueous in less concentration than hypertonic solutions. Slight traces of isotonic solution reached the aqueous. In eyes treated with testicular extract the content or sodium salicylate in the aqueous increased markedly with all three solutions. The results with potassium sulfocyanate were the same except that the isotonic solution could not be detected in the aqueous of the control eyes but was definitely present in that of eyes treated with the testicular extract. Later experiments showed that other tissue extracts, e. g., those of spleen and muscles, increased the penetration of drugs into the eye. Rossi believes that the osmotic permeability of the cells is affected by tissue extracts.

S. R. GIFFORD.

The Pupil

ISOLATED ABOLITION OF THE PUPILLARY ADAPTATION TO LIGHT (THE SEMEIOLOGICAL VALUE OF THE ARGYLL ROBERTSON SIGN). H. LAGRANGE and A. M. LAGRANGE, Ann. d'ocul. **172**: 729 (Sept.) 1935.

This article is continued from the August number, and deals with the isolated abolition of the pupillary adaptation to light in nonsyphilitic persons. The subject is treated under the following heads:

I. Generalities. The condition may be traumatic, infectious or associated with nonspecific lesions of the nerves.

II. Classification of Observations. This topic is subdivided into: 1. Abolitions of traumatic origin, which are subdivided into three groups, (*a*) those in which the lesion is connected with the encephalitic system; (*b*) those in which the lesion is ocular or orbital, and (*c*) those in which it is cervical or thoracic. Different cases from these groups are described. 2. The Argyll Robertson pupil and cerebral tumors: (*a*) generalities and (*b*) classification of observations. 3. The Argyll Robertson pupil in nontumorous peduncular lesions. 4. Abolitions of the reflex in the course of different involvements of the nervous system, such as sclerosis, syringomyelia, epidemic encephalitis, cerebrospinal meningitis and febrile diseases. 5. Abolitions of the reflex in nonsyphilitic conditions. 6. The Argyll Robertson pupil of toxic origin.

III. Adie's Disease. This topic is divided into: (1) historical considerations; (2) the syndrome, which is characterized by a pupil with slow movement (*bradycorie*); (3) a critical study of Adie's syndrome, and (4) some recent observations.

S. H. McKee.

Argyll Robertson Sign Persisting Three Years After Herpes Ophthalmicus. P. Hermann, Bull. Soc. d'opht. de Paris, April 1935, p. 226.

The patient, 58 years old, had periods of severe pain with conjunctival injection, photophobia and lacrimation. Examination revealed active inflammation of the right eye. The cornea was the site of small interstitial opacities, while the epithelium appeared irregular, stained with fluorescence, and was completely anesthetic. There was a history of a severe attack three years previously, with recurrences thereafter three or four times a year. The pupils were equal and regular and reacted promptly in convergence, but while the left reacted to direct light, the right showed no such reaction. Neither was there a consensual reaction on the affected side. The rarity of the condition is the reason for the report.

L. L. Mayer.

The Physiology and Pathology of the Pupil: II. The Course of the Normal Light Reflex. H. Machemer, Klin. Monatsbl. f. Augenh. **94:** 305 (March) 1935.

In a previous paper Machemer reported on kinematographic measuring of the pupil and registration of the motion of the pupil (*Klin. Monatsbl. f. Augenh.* **91:** 302 [Sept.] 1933; abstr., Arch. Ophth. **12:** 443 [Sept.] 1934). In this paper he reports on measuring the time required for the pupillary reflex to light to disappear. After rendering a literary synopsis in point and describing his method of examination, he shows the results and illustrates them by a number of curves and tables. The motion of the iris is analyzed, and the mathematical methods applied are given. The following conclusions are gathered as to the time required for the normal reflex to light of the same pupil to disappear:

1. The preliminary dilatation of the pupil does not represent a regular phenomenon caused by the reflex to light.

2. The duration of the latent period of the reflex of the pupil is constant. It equals 0.21 to 0.22 second and is not dependent on the original width of the pupil.

3. The reflex to light is a uniform motion during the actual period of contraction; it can be mathematically defined.

4. The initial velocity of the contraction and the velocity of the following diminution of this contraction are influenced by the initial width of the pupil.

K. L. STOLL.

Retina and Optic Nerve

SUBJECTIVE "LIGHTNING STREAKS." R. F. MOORE, Brit. J. Ophth. **19:** 545 (Oct.) 1935.

Moore calls attention to a sharp-cut symptom complex: Flashes of light, almost always compared to lightning, are seen periodically for a few weeks or a month or two and, as a rule, are referred to the temporal side of the field of vision. The flashes are most conspicuous, as would be expected, in the dark and are either accompanied or followed by an appearance of spots before the eyes.

Examinations of the fundus under mydriasis and plottings of the visual fields were made systematically lest some early organic retinal lesion, such as commencing detachment, vascular disease or neoplasm, might be the cause. Of twenty-six patients with these symptoms, twenty-one were women and five were men; the average age was 63 years, and the youngest patient was 42. The right eye was affected in nine cases, the left in thirteen and both eyes in three. In only two cases was there a history of injury.

Moore is disposed to think that the likeliest cause is the gradual increase in size and subsequent rupture of a peripheral retinal cyst, though he has no explanation to offer for either the sex incidence or the site of the phenomenon.

W. ZENTMAYER.

ANGIONEUROTIC DISTURBANCES OF THE RETINA. A. CASTELLI, Ann. di ottal. e clin. ocul. **63:** 561 (Aug.) 1935.

Castelli reports four cases of vascular disturbances which he believes should be placed in this group, all in young persons, three of whom were women. All the patients gave a history of general disturbances characteristic of the vasoneurotic type, such as profuse sweating, circulatory disturbances of the extremities, dermatographia and severe periodic headaches. In two patients definite symptoms of ovarian insufficiency were present. The first patient suddenly lost vision in both eyes. Eighteen days after the onset vision was reduced to 2/10 in the right eye and to counting fingers in the left. The fundus showed swelling of the disk and marked circumpapillary edema. The vision improved on injections of acetylcholine. Following a pharmacodynamic test with epinephrine hydrochloride given subcutaneously vision dropped rapidly to 2/50 in the right eye, which the author considered definite proof that the condition was due to vascular spasm. Subsequent use of

large doses of acetylcholine (0.4 Gm. per day) caused further improvement of vision, which finally reached normal in both eyes and remained so for a year after the onset. Another case was similar in that the loss of vision came on suddenly, vision in each eye being reduced to counting fingers, and the fundus showed similar changes. Small doses of acetylcholine were without definite effect, but when the dosage was increased to 0.3 Gm. per day there was an improvement in vision, which finally reached normal in both eyes six weeks after the onset. In another case vision was markedly reduced during short attacks and then returned to normal. Only moderate blurring of the margins of the disk was noted, with congestion of the veins. Active ovarian hormone gave marked relief from the attacks in this case. Castelli believes that the picture is due to a spasm of the vessels at some point with a slowing of the arterial current and resulting congestion. He assumes that the same condition occurs in other parts of the body but becomes manifest only as it affects the eyes. When it affects women, signs of glandular insufficiency should be looked for.

S. R. GIFFORD.

THE DIFFERENTIAL DIAGNOSIS BETWEEN CHOKED DISK AND PAPILLITIS. M. BAURMANN, Arch. f. Ophth. **134:** 189 (Aug.) 1935.

The pressure in the retinal veins as determined with Baurmann's dynamometer may be found to be abnormally increased in papilledema as well as in bilateral papillitis. In papillitis, however, the pressure in one eye is more commonly higher than in the other and the difference may within a few hours become reversed. An instructive case of bilateral papillitis is reported.

P. C. KRONFELD.

ANGIOID STREAKS. W. REICHLING, Deutsche med. Wchnschr. **61:** 1394 (Aug. 30) 1935.

Reichling observed three patients with angioid streaks in the retina. Two of these patients had a careful dermatologic examination, which, however, gave negative results. Although the presence of pseudoxanthoma elasticum could not be demonstrated, Reichling is inclined to agree with observers who consider the disorder a systemic disease of the elastic substance.

W. ZENTMAYER.

IS HYPERTENSION INSTRUMENTAL IN AIDING THE DEVELOPMENT OF DIABETIC RETINITIS? REPORT OF CASES. Y. KOYANAGI, Klin. Monatsbl. f. Augenh. **94:** 355 (March) 1935.

Volhard, in 1921, stated that he had never seen diabetic retinitis without hypertension. This view was supported by internists and ophthalmologists, cited in this paper. The contribution was prompted by the differing opinions expressed recently by Lichtwitz and Schieck. Koyanagi reports fifteen cases of diabetic retinitis and the histologic observations in one eye. In half of his cases the blood pressure remained below 150 mm. and in only five cases did it reach a pathologic height. The axiom "no diabetic retinitis without hypertension" must be abandoned therefore. Furthermore, Volhard's angiospastic theory that only the pale hypertension of over 170 mm. may explain the presence

of albuminuric retinitis cannot be applied to the origin of diabetic retinitis; this hypertension never presents a rise such as that of the so-called malign sclerosis.

In two cases spontaneous improvement occurred with transient disappearance of the retinal changes, though the patients received no systematic treatment. This fact, in Koyanagi's opinion, may be adduced as a proof against angiospastic retinal disease with malign hypertension. The prognosis of the diabetic form is far less unfavorable than that of the nephritic form: three patients with diabetic retinitis were comparatively well after six, seven and ten years, respectively.

Clinical observation of one case, the sixth, allowed no conclusion as to whether the diabetic retinitis was complicated by hypertension or by nephritic disorders. The histologic examination proved that the diabetic retinitis had developed as an independent disease. Edema of the retina was absent histologically, and it had never been observed with the ophthalmoscope. The white retinal foci, also noted clinically, consisted of transudates abounding in albumin but free from fibrinous masses; the transudation was well defined and did not spread into the surroundings. Lipoid substances, proliferation of the neuroglia, deposits of fibrin and varicose hypertrophy of the nerve fibers were absent. These white spots, unlike those found in nephritic retinitis, are of no consequence in diabetic retinitis. The pigment layer was perfectly normal.

K. L. Stoll.

The Obscure Influence of General Blood Pressure on the Progress of Optic Nerve Disease. H. Lauber, Wien. klin. Wchnschr. **48:** 1079 (Aug. 30) 1935.

From the investigations of Bailliart, Baurmann and especially Sobański, Lauber is led to believe that the existence of choked disk depends on a loss in the normal relationship between the diastolic pressure in the retinal vein and that in the artery. If the general blood pressure is high the relation of the diastolic venous pressure to the diastolic arterial pressure is within normal limits; that is, it does not fall below 1 to 1.9. Thus a choked disk is not produced under such a circumstance even if the intracranial pressure is very high. But if the general blood pressure is proportionately lower—that is, not increased above the normal—the diastolic pressure is changed so that a relationship of 1 to 1.5 is in evidence; then choked disk ensues.

This choking appears the more quickly the more the diastolic pressure is lowered. The intra-ocular pressure also plays a rôle in that its pressure varies with the influence of the choked disk. From this premise Sobański has been able to explain pseudoneuritis. The intra-ocular pressure in these patients is low. In general, the comparative visual acuity of an eye is likewise dependent on the relationship between the general blood pressure and the retinal arterial tension. This is more evident in disease of the optic nerve, for example, tabes. Magitot has used this principle in operating on the carotid sympathetic nerves to increase general blood pressure to the eye. Further studies are necessary to draw more definite conclusions, but the importance of the relationship is firmly established.

L. L. Mayer.

Trachoma

THE PRECISE ORIGIN OF CORNEAL PITTING. H. HERBERT, Brit. J. Ophth. **19:** 600 (Nov.) 1935.

Herbert regrets that it has been deduced from his remarks that he is "not in agreement with the view that follicles are the antecedents of Herbert's pits." He states that all the more obvious marginal post-trachomatous corneal depressions, whether typically follicular in the arrangement of the cells or not, indicate the sites of former cell aggregations.

W. ZENTMAYER.

RICKETTSIA-LIKE BODIES IN TRACHOMA FOLLICLES. A. CUÉNOD and R. NATAF, Rev. internat. du trachome **12:** 110 (July) 1935.

The inclusion bodies considered by Thygeson as related to the etiologic organism of trachoma have not been found in the subepithelial tissue or in the interior of the trachoma follicle. However, confirming the previously published work of Busacca, the authors have found in these areas rickettsia-like bodies constantly and in large numbers. These bodies are subbacterial in size and morphologically similar to the rickettsiae observed in typhus, trench fever and tick fever.

J. E. LEBENSOHN.

TRACHOMA FOLLICLES IN THE CHOROID AND IN THE VITREOUS OF ANIMALS AFTER INTRA-OCULAR INOCULATION WITH TRACHOMATOUS MATERIAL. A. VON SZILY, Klin. Monatsbl. f. Augenh. **94:** 320 (March) 1935.

In a previous publication (*Klin. Monatsbl. f. Augenh.* **94:** 1 [Jan.] 1935; abstr., ARCH. OPHTH. **15:** 147 [Jan.] 1936) von Szily reported on his experiments in inoculating rabbits' eyes with trachomatous material. This paper describes further experiments, which corroborated the conclusions drawn: Typical trachoma follicles developed in the eyes, especially in the choroid and vitreous. The results of control tests with nontrachomatous material were negative. Photomicrographs show solitary follicles and groups of them in the choroid, the groups appearing chiefly where the suprachoroid was rarefied by advanced shrinking of the eyeballs. They occurred partly in the choroid directly below the pigment layer and partly between the rarefied tissues of the suprachoroid. Some of these follicles reached considerable size; again, two or three of them as they grew larger became united. The size and demarcation of the follicles depended on the density of the surrounding tissues. Solitary follicles were present in large number in the fine scar tissue that formed secondarily in the vitreous. They arose about three to four weeks after the inoculation. They were less well defined than those in the choroid and were surrounded by a denser wall of lymphocytes than surrounded the follicles in the choroid. The cell elements produced by the virus were derived from the blood vessels: in the choroid, from those blood vessels present and, in the vitreous, from those formed by the primary inflammation. Minute nodules with active mitosis developed around the canal of inoculation of the virus; they represented the stratum out of which the follicles developed.

K. L. STOLL.

Tumors

A Case of Endothelioma of the Optic Nerve Sheaths. H. B. Stallard, Brit. J. Ophth. **19:** 576 (Oct.) 1935.

The patient was a man aged 30. There was a history of hazy vision for five days. Vision was reduced to perception of hand movements in the temporal field. Two thirds of the optic disk on the nasal side was edematous, and the edema extended into the retina. Eighteen months later new-formed vessels were present on the anterior surface of the iris. There was an extensive detachment of the retina as well as a dark area adjacent to the disk. There was secondary glaucoma. Retinal hemorrhages were present. The eye was blind. The eye was enucleated. The diameter of the nerve 16 mm. behind the globe was 17 mm. The neoplasm was pyriform. The dura was thickened. The arachnoid occupied by the growth completely surrounded the nerve and compressed it to a thin cord. The neoplasm had a yellowish granular appearance. A homogeneous exudate occupied the interretinal space.

The tumor cells were oval-shaped, endothelial in type, with a granular cytoplasm and a nucleus with a well defined chromatin network and nucleoli. The cell masses were broken up by vascularized connective tissue trabeculae and collagenous fibrils. The optic nerve was atrophic and showed hyaline degeneration. A branch of the retinal vein was thrombosed. The filtration angle was occluded by peripheral synechiae.

The article is illustrated.

W. Zentmayer.

Mixed Tumor of the Lacrimal Gland. Hoche, Baudot, Gault and Legait, Bull. Soc. d'opht. de Paris, April 1935, p. 249.

Neoplasms of the lacrimal glands are exceptional and descriptions of them in the literature sparse. One fact is well established, that a large proportion, fully 85 per cent, of the neoplasms reported are of the mixed type. Adenoma and epithelioma of the lacrimal gland are exceedingly rare. A brief review of the various types of lacrimal tumors precedes the authors' review of their case. A patient, aged 59, had a small lump near the external angle of the right eye for approximately ten years. From time to time symptoms such as fulness, swelling and pain occurred but were of slight nature. Recently these were exaggerated, and an exophthalmos of 34 mm. developed. The tumor was not adherent to the globe, nor did it interfere with ocular motion. The mass was removed under local anesthesia. It was not adherent to the orbit. It was surrounded by a fibrinous capsule and directly connected with the lacrimal gland. It measured in the neighborhood of 3.5 cm. in each of the three directions. A mucous or myoid tissue replaced the glands, surrounded by the cuboid and cylindric cells of the tubules. Fibrous tissue, hyaline cartilage and disseminated epithelial cells were found well mixed in many sections. Involvement of the cornea was not noted, and this was rarely reported in previously discussed cases. The tumor is refractive to roentgen therapy, and surgical intervention is always advisable. A photograph of the patient and two photomicrographs of the tumor are shown.

L. L. Mayer.

FAMILIAL NEUROFIBROMATOSIS. K. T. A. HALBERTSMA, Arch. f. Ophth. **134:** 167 (Aug.) 1935.

Halbertsma reviews the literature concerning ocular manifestations of neurofibromatosis (glaucoma, degeneration of the optic nerve, blastoma of the retina or of the optic nerve, neuroma of the lid) and reports the pedigree of a family in which three members of one generation and one member of the following generation were affected. The inheritance seemed to be, except for some irregularities, of the dominant type.

P. C. KRONFELD.

Uvea

NONSURGICAL REATTACHMENT OF IRIS IN A CASE OF TRAUMATIC IRIDODIALYSIS. V. J. SCHWARTZ, J. A. M. A. **105:** 1032 (Sept. 28) 1935.

A boy, aged 12 years, was injured in the left eye by a torpedo. The iris was torn loose from its attachment superior to the ciliary body for a distance of 6 or 7 mm. There were accompanying hyphemia, hemorrhage into the vitreous and rupture of the lens capsule.

Under rest and mydriatics only, the iris became completely reattached where it had been torn away from the ciliary body and has remained so to date (a period of six years). Cataract developed and was successfully removed by needling.

W. ZENTMAYER.

ASPECTS OF HEERFORDT'S SYNDROME (UVEOPAROTID FEVER). M. HAFERKORN, Monatschr. f. Kinderh. **63:** 161 (July 20) 1935.

Haferkorn points out that Heerfordt's syndrome, in which fever, iridocyclitis and parotitis are the most noteworthy symptoms, occurs mostly in children and young adults. He describes a case of his own observation. In a boy, aged 13, there developed in rapid succession paralysis of the three branches of the facial nerve, swelling of the parotid gland and iridocyclitis. The patient also had polydipsia and polyuria. He thinks that the polydipsia was the result of a deficient secretion of the parotid gland, for diabetes insipidus could be excluded on the basis of the inefficacy of hypophyseal preparations and the result of the sodium chloride tolerance test. The swellings of the parotid gland and of the bronchial lymph nodes yielded to roentgen therapy. In discussing the etiology of disease of the uveoparotid gland Haferkorn points out that Heerfordt assumed an infectious-toxic origin and rejected a connection with mumps. However, several other investigators gave consideration to the problem of chronic mumps, for in a number of cases including the reported case an attack of mumps preceded the development of the syndrome. Other authors assume a connection with tuberculosis. In the few cases in which a histologic examination of the eyes was made the diagnosis of tuberculosis was corroborated. In the author's case roentgenologic examination of the lungs revealed signs indicative of tuberculosis of the bronchial lymph nodes. Moreover, a calcified tuberculous focus was found in the abdomen, and an excised piece of the parotid gland showed caseous granulation tissue, which doubtless was of tuberculous character.

J. A. M. A. (W. ZENTMAYER.)

Visual Tracts and Fields

INVESTIGATION OF THE VISUAL FIELD AT LOW OXYGEN AND ATMOSPHERIC PRESSURES. W. KYRIELEIS, A. KYRIELEIS and P. SIEGERT, Arch. f. Augenh. **109:** 178, 1935.

The authors repeat the work of Berens and of Goldmann and Schubert on mapping the visual fields in normal persons under low tension of oxygen and at reduced barometric pressure. The oxygen tension was reduced as low as 5 per cent, and the subjects were studied under a barometric pressure corresponding to a height of 8,000 meters above sea level. Under reduced oxygen tension no certain changes in the visual fields could be found, but there was a slight tendency for the fields to be concentrically widened at the beginning of the anoxemia. No changes in the visual fields occurred under reduced barometric pressure. One of the subjects had also been used by Goldmann and Schubert, who found a contraction of the nasal field under reduced barometric pressure. They explain the differences between their results and those of Goldmann and Schubert on the basis of the different methods employed.

F. H. ADLER.

IS THERE A PHYSIOLOGIC WEAKNESS OF ATTENTION IN THE NASAL PERIPHERY OF THE VISUAL FIELD? WERNER KYRIELEIS, Arch. f. Augenh. **109:** 190, 1935.

In a previous article (Kyrieleis and Siegert: *Arch. f. Augenh.* **109:** 178, 1935) the author stated that there is a definite lowering of attention when a person is subjected to the effects of low barometric pressure and anoxemia which often leads to errors in mapping the extent of the visual fields. This factor is more apt to be effective in causing constriction of the nasal than of any other portion of the visual field.

The present article is a discussion of the cause of this phenomenon. When a bright object is brought into the binocular field of vision there is a noticeable increase in brightness at the moment at which the object moves into the visual field of the second eye; in other words, when it is seen with the two eyes together. Theoretically the point of this change should occur exactly at the nasal border of the visual field of the second eye. Kyrieleis found, however, that almost regularly the point at which the object increased in brightness when seen with the two eyes was 5 degrees or more outside the nasal limits of the single monocular field. From this he concludes that the constriction of the nasal field is due to a weakness in the power of attention.

F. H. ADLER.

Vitreous

THE VITREOUS: PARTS III AND IV. F. GOEDBLOED, Arch. f. Ophth. **134:** 146 (Aug.) 1935.

An interesting though highly technical paper is this, from which one can learn that the conception of the vitreous being a "simple" gel is much too diagrammatic. In previous papers Goedbloed presented data

which indicate that the ultramicroscopic threads of the vitreous represent colloidal structures since they have been found to carry electrical charges and bound water (state of hydration). These threads have also been shown (Robertson and Duke-Elder) to be elastic, which is another characteristic of the colloidal state. The vitreous as a whole, however, possesses—in vitro—neither elasticity nor tendency to maintain its volume. The slightest pressure makes it lose water, which it does not take up again when the pressure ceases. Any kind of manipulation decreases the volume of the vitreous irreversibly, a phenomenon which has been interpreted by Baurmann as a sign of maximum turgescence under physiologic conditions and relative deturgescence under any nonphysiologic conditions.

In 1934 Baurmann for the first time succeeded in making the vitreous swell beyond its original volume by leaving it in alcohol of concentrations from 10 to 30 per cent for several days. This phenomenon has been analyzed in great detail by Goedbloed. He concludes now that even this swelling of the vitreous is principally different from the behavior of true gels because the degree of hydration of the vitreous does not increase under the influence of alcohol in these low concentrations. Furthermore, no measurable turgescence pressure is developed during the swelling. "The vitreous, under physiologic conditions, does not possess the properties of a true gel."

The second part of the paper deals with the question: How does the vitreous in vivo maintain its volume and how does it manage to withstand the intra-ocular pressure which acts on it constantly? Under experimental conditions, a pressure much lower than the intra-ocular tension makes the vitreous lose water and shrink. Goedbloed introduces the term "tonus of the vitreous," which is the pressure prevailing in vivo in the space inside the hyaloid membrane. He believes this tonus is produced by the hydrostatic pressure of the fluid contained in the meshwork of the vitreous. This tonus must be about the same as the intra-ocular pressure. The hyaloid membrane is certainly permeable to water. To maintain a constant tonus it is therefore necessary that the outflow of fluid from the vitreous should be balanced by the inflow. The inflow probably takes place at the base of the vitreous, that is, from the ciliary body as in the production of aqueous. The outflow probably takes place where there is no inflow, namely, everywhere except over the ciliary body. According to this conception a constant stream of intra-ocular fluid enters the vitreous at its base and leaves it through the retinal vessels or through the anterior channels of exit of the intra-ocular fluid. Ceteris paribus, the tonus of the vitreous should depend on the resistance which the intra-ocular fluid encounters going through it. In an increased internal vitreous resistance Goedbloed sees a possibility of explaining the mechanics of glaucoma simplex. As far as I can see, an increased resistance would only slow down the through-and-through circulation in the vitreous and would not necessarily raise the tonus of the vitreous unless one assumes that the ciliary body constantly and irrespective of circumstances produces the same amount of fluid.

P. C. KRONFELD.

Sympathetic Ophthalmia

A Simple Experiment to Prove the Tuberculotoxic Origin of Sympathetic Ophthalmia. H. Guillery, Arch. f. Augenh. **109:** 139, 1935.

Guillery gives further evidence (for full bibliography see the article) that sympathetic ophthalmia is due to the presence of tubercle bacilli. He considers sympathetic ophthalmia not an active tuberculosis but an effect produced by certain poisons from tubercle bacilli. He discusses the results of injections of phosphatide from tubercle bacilli produced by the method of Anderson and his co-workers. The effect of trauma is also discussed.

F. H. Adler.

The Inflammatory Effect of Phosphatide and Its Relation to Sympathetic Ophthalmia. Augusto Lopes de Andrade, Arch. f. Augenh. **109:** 235, 1935.

Guillery (*Arch. f. Augenh.* **106:** 359, 1932) bases his theory of the tuberculotoxic origin of sympathetic ophthalmia on experiments which he performed with the phosphatide obtained from tubercle bacilli by the method of Anderson and his co-workers. He believes that this phosphatide is the cause of sympathetic ophthalmia because he was able to produce a histologic and clinical picture of the disease as it occurs in man by injecting the phosphatide into rabbits. He assumes that no substance other than the phosphatide from tubercle bacilli can produce such a typical picture of infiltration with lymphocytes and giant cells.

The present author shows that this assumption is incorrect, and that the histologic picture which Guillery believes is pathognomonic of sympathetic ophthalmia may be produced by other noxious agents besides the phosphatide of tubercle bacilli. He injected a fluid phosphatide obtained from plants, phytin, which naturally has no relation to the phosphatide obtained from the tubercle bacilli, and obtained a histologic picture which, he claims, resembled that described by Guillery. He argues from this that the latter's assumption that sympathetic ophthalmia is a tuberculotoxic reaction is not proved.

F. H. Adler.

Treatment of Sympathetic Ophthalmia by Speransky's Method. A. T. Rumyantseva, Sovet. vestnik oftal. **7:** 27, 1935.

This method consists in the blockade of the sympathetic nervous system of the lumbar region with injections of a 0.25 per cent solution of procaine hydrochloride into the interfascial space of the kidney. On the basis of experimental and clinical data, Speransky believes that the nerve-trophic component plays an important rôle in the development of the pathologic process. The procaine hydrochloride blockade of the lumbar region is applied as a weak stimulator to distant separate foci of inflammation as a favorable therapeutic measure. In severe injuries of the ciliary body, the chief function of which is the nutrition of the eye, permanent changes occur in the eye and eventually the other eye is involved. In order to sever the connection with the central and sympathetic nervous system in sympathetic inflammation Speransky advises a temporary isolation of a nerve section for the formation of a

new internerve correlation; during this blocking a permanent disintegration of separate diseased foci will take place.

Two patients with acute and four with chronic recurring sympathetic ophthalmia were treated with the procaine blockade. In two cases of acute sympathetic ophthalmia, in which enucleation of the exciting eye and general and local treatment of the sympathizing eye were of no avail, one or two injections of procaine hydrochloride into the capsule of the kidney of the corresponding side gave a remarkable effect; ciliary injection, precipitates and synechiae disappeared within one month; the vision was improved in one case from 0.2 to 1 and in the other from 0.06 to 0.9. Three cases of chronic sympathetic ophthalmia gave only moderate improvement, and in one case the disease progressed and the vision was nil. Rumyantseva recommends further observation of the procaine blockade, as the results are particularly favorable in acute sympathetic ophthalmia.

O. SITCHEVSKA.

Therapeutics

TREATMENT OF ANGIOMA OF THE EYELID BY SCLEROSING INJECTIONS. L. WEEKERS and S. LAPIERRE, Arch. d'opht. **52:** 14 (Jan.) 1935.

The authors point out that because of the proximity of the eyelid to the eye angioma of the lid is difficult to treat. This difficulty is still greater when the angioma involves the margin of the lid, because of the possibility of deformity from scar tissue. The methods available for the treatment of angioma are briefly stated, and a more extended review of the development of treatment with sclerosing injections is presented. The technic of this treatment is described. Quinine and urea hydrochloride is used in a 7.5 per cent solution for general purposes. This is changed to a 5 per cent solution for superficial, and to a 10 per cent for thick tuberous, angioma. The use of carbon dioxide snow is also described. The principal indication for the use of sclerosing injections is the deep thick angioma. When the angioma is partly superficial and partly deep the sclerosing injections are used for the deep part, followed later by the application of carbon dioxide snow. For small superficial angioma carbon dioxide snow is used. For extensive flat angioma no treatment is used. As the result of the development of the treatment by sclerosing injections the authors feel that the indications for the use of radium and the x-rays are diminishing.

S. B. MARLOW.

Toxic Amblyopia

NYCTALOPIA CAUSED BY TOBACCO AMBLYOPIA IN CHAUFFEURS. A. FUCHS, Klin. Monatsbl. f. Augenh. **94:** 377 (March) 1935.

Fuchs adds one case of nyctalopia with tobacco amblyopia in a chauffeur to Rollet's two cases of hemeralopia in chauffeurs. A chauffeur, aged 45, complained of impaired vision for a month. Vision was reduced to 5/36, the disks were normal, and an oval scotoma for red existed, extending from the point of fixation to the blindspot. Vision was almost normal after two months' treatment with hypodermic injections of strychnine. This man drove his auto very slowly during the day and seemed not to see pedestrians, although the peripheries of his

fields were normal. In the dusk, however, and as soon as the street lamps were lighted, he drove rapidly and reliably. No change of the vision was noted under varying strengths of the illumination of the test charts.

K. L. STOLL.

TOXIC ACTION OF TRICHLORETHYLENE ON THE EYE: REPORT OF A CASE. E. KUNZ and R. ISENSCHMID, Klin. Monatsbl. f. Augenh. **94:** 577 (May) 1935.

Trichlorethylene (CCl_2-CHCl) is used in several branches of manufacture, e. g., in the metal industry to cleanse metals, and, furthermore, to extract oil from seeds, wool, leather and skins. It is contained in preparations to exterminate parasites and in shampoos. It is employed to dissolve rubber, tar, rosins, and pitch, also to prevent rust, and to paint the inside of beer barrels.

A man, aged 56, used trichlorethylene in grinding diamond to powder and to remove this powder from the grinding wheels. Chronic poisoning resulted, with retrobulbar neuritis, polyneuritis and rigidity of the pupil to light. Cases known through the literature show that the optic nerve was usually affected by the toxicity of this preparation. Other ocular symptoms may be incidental. This type of poisoning can no longer be classed with the toxic amblyopias combined with various functional disturbances. It is related rather to alcohol amblyopia but is of a more unfavorable prognosis.

K. L. STOLL.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

AMERICAN OPHTHALMOLOGICAL SOCIETY

Seventy-First Annual Meeting, June 5, 6 and 7, 1935

(*Tr. Am. Ophth. Soc.* **33**:26, 1935)

WALTER B. LANCASTER, M.D., *President*

J. MILTON GRISCOM, M.D., *Secretary*

DIAGNOSIS AND DIFFERENTIATION OF TYPES OF ALTERNATING CONCOMITANT SQUINT. DR. LUTHER C. PETER, Philadelphia.

The increased interest in the correction of amblyopia ex anopsia and especially in the restoration of single binocular vision in patients with monocular squint together with similar results said to have been obtained in those with alternating strabismus lead me to believe that a discussion of the types of alternating concomitant squint may be of value.

For comparison the accompanying table affords a summary of the chief points of difference in monocular, accidentally alternating and essentially alternating squint.

Points of Difference in Types of Alternating Concomitant Squint

Point of Comparison	Monocular Squint	Accidentally Alternating Squint	Essentially Alternating Squint
Age of onset	One to four years	One to four years	At birth or soon after
Amblyopia	Marked	Slight and promptly corrected	None
Refraction	Usually marked hyperopia	Usually marked hyperopia	Slight to moderate hyperopia
Anisometropia	Often marked	Rarely marked	None
Fusion	Present but weak	Present but weak	Totally absent
Results of fusion training	Good	Good	Nil

The importance of differentiating clearly between the two types of alternating squint becomes obvious in orthoptic training. Accidentally alternating squint yields readily to orthoptic training and single binocular vision with stereopsis is obtainable if one follows the same methods as in the treatment of monocular squint. In contrast, in true alternating strabismus fusion training not only fails but causes serious damage through the more or less imperfect breaking up of the habit of suppression—a habit that is necessary to the comfort of the patient. I have never seen a case of true alternating squint in which fusion could be developed. Furthermore, simultaneous macular perception cannot be

obtained so long as suppression is as highly developed as it is with this type of squint.

If each case is studied with sufficient care, the observer rarely fails to make the proper classification. When doubt arises because the characteristics of a given case are not clearly differentiated, the presence of simultaneous macular perception will help to answer the question. In addition, if even weak fusion can be demonstrated on the synoptophore and if such fusion can be strengthened, the evidence is conclusive that an accidentally alternating deviation exists. The absence of simultaneous macular perception and of fusion is characteristic of true alternating squint.

DISCUSSION

DR. J. H. DUNNINGTON, New York: Worth's classification of cases of squint as outlined by Dr. Peter is still much debated, but, as he has aptly said, only by carefully analyzing one's cases can one treat strabismus intelligently. Very important in such an analysis is full information as to the presence or absence of simultaneous macular perception and of fusion power. All ophthalmologists are agreed as to the value of this information, but some are not in accord with the author as to the management of the small group of patients in whom there is complete absence of fusion and of simultaneous macular perception. My experience is not sufficiently large to allow me to enter into the discussion except to say that in most of the cases there will be a resort to operation and the result will be a purely cosmetic one. It has not been my practice to consider only the squint with complete absence of fusion as true alternating squint, but regardless of what one calls it I am sure Dr. Peter is correct in the management of patients of this class.

It seems to me that the important lesson to be learned from this presentation is that, in the complete absence of simultaneous macular perception and of fusion, orthoptic training is not only futile but at times even harmful. One is living in an age in which it is the vogue to treat all ailments by physical methods, so that one must be careful not to let enthusiasm overshadow sound judgment. This is particularly true in relation to orthoptic treatment, which should be instituted only after a most careful analysis of the clinical findings. One must remember that some patients will be more uncomfortable with defective fusion than with complete suppression.

DR. CONRAD BERENS, New York: This is so important a subject that a few cases in which squint was apparently associated with aniseikonia are reported for Dr. Peter's criticism. Well marked aniseikonia was found in 5 patients who had alternating esotropia (that is, they alternated fixation, with no preference for fixation with either eye, and had substantially equal visual acuity in each eye). (The findings are presented in tabular form.)

Findings of this character lead me to believe that the whole question of strabismus, especially of alternating strabismus, may require restudy in the light of the improved knowledge of aniseikonia. At the present time the technic is far too complicated to be applied in practice, except in patients who have fair second grade binocular vision.

DR. LUTHER C. PETER, Philadelphia: The members are indebted to Dr. Berens for injecting the question of a difference in size into this problem. It is a matter with which one will have to reckon in the future. I am sure that it will often be found a factor in abnormal motility of the eye. With respect to the rôle that aniseikonia plays in squint, I am of the opinion that it falls under accidental alternating strabismus. It causes an impasse which prevents the patient from fusing, and when the difference in size is corrected, if the fusion faculty is normal, there should be perfect fusion. From my experience over a number of years I do not believe that any true alternating squint can be cured and the power of fusion restored. I shall be very glad to hear from any one who has cured such a squint. Worth, in his book, is on record in a number of instances as to the incurability of true alternating squint, and my experience is entirely in accord with his views.

DIATHERMIC AND GALVANIC TREATMENT OF SEPARATED RETINA, ESPECIALLY OF THE FLAT TYPE, WITH SPECIALLY DEVISED MINIMAL STOP AND SINGLE AND MULTIPLE PLATINUM MICRO-ELECTRODES. NEW MAGAZINE CONTAINERS. DR. CLIFFORD B. WALKER, Los Angeles.

An attempt to evaluate the possibilities in closure of retinal tears by the use of electrolytic alkali applied by catholysis as described by Vogt has confirmed my statements made after 15 operations by a modified Vogt method in practically every detail.

Every type of case, described by Vogt (except the case illustrated by figure 6) has been observed, and 3 of separation of the retina, in which the latter was perhaps more extensively damaged than in any included in his report, are briefly recorded here.

The upper limit of treatment with alkali as applied by catholysis was not ascertained, although it was believed that a record was set when 126 punctures, each of about one second of terminal duration, were made with a needle having a diameter of 0.006 inch (0.015 cm.), carrying on the average 0.85 milliampere of current and penetrating 7 mm., with every evidence of success. Also, 0.02 cc. of 3 per cent solution of potassium hydroxide was injected subchoroidally at the macular region in the same eye.

However, failure occurred in a case with a traumatically torn hole in the retina from two to three times the size cited by Vogt, extending centrally 45 degrees from the macula. But in 2 cases, one of myopia and the other of trauma, a large 70 to 75 degree dialysis below the macula, also with a macular hole, was reattached, with excellent restoration of visual field and vision.

Indications point to the fact that perforation of the retina is not necessary in all cases, although it is often unavoidable with this method of treatment.

From the position of an accessory to diathermic treatment, alkali therapy in some form, even that by a hypodermic microneedle, may easily come to occupy the more important position in the treatment of separated retina.

For the details of the technic, the original paper should be read (*Tr. Am. Ophth. Soc.* 33: 48, 1935).

DISCUSSION

DR. MARK J. SCHOENBERG, New York: It would not be wise to give up the diathermic treatment in favor of the Vogt method until a little more is known about the latter. One objection to his method is the possibility of damage to the retina. I do not believe that puncture of the retina is entirely harmless. Another difficulty is that the punctures, being very fine, are not visible on the surface of the sclera. One must use a microscope or a very strong magnifying glass to see them, and if one is not certain that a given area has been thoroughly punctured, there is no way of knowing which spots must be treated again.

So far as the hook electrode is concerned, I have found it very satisfactory. The advantage is that it acts as a retractor, pulling the eyeball forward at the same time that the current is applied. Being oblique and hooklike, it penetrates the sclera in an oblique direction, and with a little experience one can estimate the distance it must go through the sclera to reach the choroid without perforating the retina. If the detachment is deep, one does not need to worry about injuring the retina; but if it is shallow, one must be careful not to send the needle in too deep.

I also have found that local anesthesia blurs the cornea, thereby preventing a thorough examination of the fundus during the operation. For that reason I have discarded the use of anesthesia obtained by drops. I do not generally use a speculum in order to prevent trauma of the corneal epithelium.

DR. ALEXANDER E. MACDONALD, Toronto, Canada: It seems to me that the simpler the procedure which is used the better are the results. At the Toronto General Hospital the Moorfield modification of the Larsson technic has been found extremely satisfactory. It appeals to me for the reason that one obtains an area of reaction and that less damage is done to the choroid and the retina. One obtains an electrochemical reaction, and each point is localized; the retina is sealed down by the inflammation that ensues. None of the perforating methods appears to me to be efficient. I looked over a series of eyes and found choroidal hemorrhages present in over 30 per cent, which indicates that choroidal hemorrhages are much more frequent than has hitherto been believed. I am of the opinion that any perforating method adds to the danger of hemorrhage.

DR. WILLIAM H. CRISP, Denver: It may be worth mentioning that Bietti, an Italian clinician, has recommended a special technic, that of using carbon dioxide snow. He mixes the carbon dioxide snow with acetone in a specially devised syringe, which has a curved tip with a very fine end. He claims a number of excellent results. The carbon dioxide mixture is entirely enclosed within the syringe and tip and is not applied directly to the eyeball but produces the freezing effect through the metal. The application is made to the outside of the sclera. It causes an inflammatory reaction with the formation of a subsequent retinochoroidal adhesion. The method appears to be somewhat simple and well worth attention.

DR. F. H. VERHOEFF, Boston: I have been interested in the fact that Vogt has recently advocated the treatment of separation of the retina by electrolytic punctures. In 1916 I published a description of

a similar method. I believe that that was the first time that any one suggested using multiple punctures of the sclera for fastening down the retina. At that time, I used electrolysis, so that I am interested in hearing that the electrolytic method is again being advocated, although it is now termed the Vogt method.

The injection of fluid into the vitreous after the scleral punctures have been made has been employed by others but not, I believe, in the same way in which I have employed it. After making the scleral punctures and before the needles are removed, I insert a short fine hypodermic needle into the vitreous through the sclera and pars plana of the ciliary body. Attached to the needle is a long stretch of fine rubber tubing connected with a large syringe without a plunger. The syringe and tubing are filled with half-strength physiologic solution of sodium chloride. Before the needle is inserted, the sclera is almost perforated by pricking it with the point of a cataract knife so that the needle can be pushed through with very little force. The syringe is elevated so that a pressure of at least 20 mm. of mercury is obtained. The puncture needles are then removed, and the fluid is allowed to run into the eye until the flow ceases. Fluid can be seen coming out of the puncture holes. I used this method many years ago, but it failed, evidently because there was so large a hole in the retina that the fluid ran out without replacing the retina. It is evident that if the fluid fails to cease flowing, the retina cannot be replaced, and the case is hopeless. In 4 recent consecutive cases the method was entirely successful. Whether the distention of the vitreous with fluid will ultimately prove to be advantageous or harmful cannot be decided by theoretical considerations, and I shall therefore continue to use the method until I obtain evidence that it is harmful. Its advantage is that it replaces the retina completely and at once.

Dr. Clifford B. Walker, Los Angeles: I may say with regard to surface treatments of all sorts that in my hands they have been able to produce only a small number of cures at the expense of considerable reaction and external scar formation. Vogt regards all methods of surface treatment only as accessory to the best methods of penetrating treatment. It appears, for the most part, that the damage to the sclera in surface treatment is disproportionately greater than is necessary to produce the desired amount of chorioretinitis by certain other methods. The surface treatment seems to have only the advantage of being considerably easier to carry out, and the effect is possibly somewhat less hemorrhagic, but, on the other hand, the reaction is severe and iridocyclitis and cataract formation counterbalance the advantage. Secondary operations are much more difficult to perform after surface coagulation than after galvanic puncture or micropuncture. Cures may be obtained by almost all the methods that have been reported, but I agree with Dr. MacDonald that one must choose the method and the technic that give the highest percentage of lasting good results.

Dr. Verhoeff's method of hydrostatic replacement is daring and ingenious, but it again must stand on the percentage of good results obtainable over the course of time. My own theory as to what is the method of maximum efficiency seems to be that it is a combination of micropuncture with some form of alkali treatment with electrolytic or

direct chemical application. I do not believe that a single method will show maximum efficiency with all types of retinal separation.

Dr. Schoenberg's question concerning the application of the positive pole is answered fully in the paper. Briefly I may say that I modify Vogt's technic by raising the voltage of the current to 45, so that the regular diathermic pad can be used in applying the positive pole on the shoulder as with diathermy. When the diathermic pad is connected in, a three-way switch is used to throw the pad from the galvanic unit to the diathermic unit and vice versa, with one turn of the switch.

SUBJECTIVE STUDIES OF THE BLIND SPOT AND VISUAL FIELDS. DR. EDWARD JACKSON, Denver.

This paper was published in full in the *American Journal of Ophthalmology* **19**: 34 [Jan.] 1936.

THE CENTRAL VISUAL FIELDS AND PUPILS IN PATIENTS WITH NEUROSYPHILIS. DR. C. W. RUTHERFORD, Iowa City.

Neurosyphilis was diagnosed in 93 patients who were admitted to the department of neurology of the University Hospital in 1934. Dementia paralytica was diagnosed in 20 patients, tabes in 30, meningovascular syphilis in 36 and mixed types in 7. In the department of ophthalmology these patients were examined with respect to visual acuity and the state of the pupils, fundi and central visual fields.

Argyll Robertson pupils were found in 59 eyes. In only 1 patient was the phenomenon unilateral. In 5 of these eyes the pupils were oval, and in 5 they were irregular in shape.

Anisocoria was present in 16 patients.

The visual fields were apparently not influenced by the age of the patient, the central visual acuity or the size of the pupil. The average extent of the fields did not help to differentiate among the several types of neurosyphilis.

Contracted visual fields were observed in 88 per cent of the patients and in 84 per cent of the eyes. A superior contraction was found in 69 per cent, a nasal in 49 per cent, and a combined superior-nasal in 41 per cent, of the fields.

The blind spots were enlarged in 31 per cent of the fields, and, on the average, fields with enlarged blind spots were much more contracted than those without enlarged blind spots. Enlarged blind spots and superior-nasal contractions were associated in 15 fields.

The study yielded the following observations: 1. Argyll Robertson pupils did not alone account for the field contractions. 2. The fields in eyes with oval or irregular pupils were more contracted than those in their fellow eyes with round pupils. 3. Contractions in the fields, especially of the superior-nasal quadrants, were prominent. 4. Extensive contractions were found in association with enlarged blind spots. 5. The most severe contractions occurred with atrophy of the optic nerve.

DISCUSSION

DR. ALBERT N. LEMOINE, Kansas City, Mo.: To depend entirely on the laboratory for the diagnosis of neurosyphilis would be disastrous.

This was well brought out in my study of congenital syphilis. Certain children of parents known to be syphilitic gave negative results in serologic tests and later showed signs pathognomonic of congenital syphilis. Even the spinal fluid of a few of these children gave negative reactions to all known tests. Syphilitic lesions of the eye were more frequent and constant than lesions of other parts of the body.

HOMONYMOUS HEMIANOPIA: SOME PRACTICAL POINTS IN ITS INTERPRETATION, WITH A REPORT OF FORTY-EIGHT CASES IN WHICH THE LESION IN THE BRAIN WAS VERIFIED. DR. THOMAS H. JOHNSON, New York.

This paper will be published in full with the discussion in a future issue of the ARCHIVES.

NEW AND PRACTICAL CHARTS AND LIGHTING FOR TESTING VISUAL ACUITY AND LOCATING THE ASTIGMATIC AXIS. DR. M. HAYWARD POST, St. Louis.

The charts are built on the principle of rotating dials carrying the test objects and surmounted by a rotating shutter the function of which is to expose the object or objects desired while excluding all others. The suitability of the broken ring of Landolt as a standard test of visual acuity is demonstrated, also that of a more complex figure, such as the Maltese cross of Beach, for accurate testing of refraction. The value of the double broken ring of Landolt as modified by Ferree and Rand for the analysis of astigmatism is pointed out. Attention is called to the great importance of a variable standardized illumination for all tests of visual acuity, and simple lamps for securing such lighting are described.

DISCUSSION

DR. S. J. BEACH, Portland, Me.: One somewhat frequently finds that distortion of the refractive media has resulted in irregular axes. Astigmatic dials are then deceptive, especially if they are the half-dials, like the astigmatic fan. Under such circumstances the broken circles, whether they have one or two breaks in them, are practically valueless. This is one of the reasons why ophthalmologists probably will never entirely dispense with the ordinary Snellen chart. I therefore suggest that letters or symbols with more acute angles be incorporated. This apparatus has the merit of enabling the examiner to use the symbols which he prefers and which he finds most useful. It should be a valuable addition to the admamentarium.

DR. ALFRED COWAN, Philadelphia: For subjective studies of refraction and the determination of the axis of astigmatism charts in a great variety are available, among the best of which should be mentioned those of Lancaster, Beach and Green, the Verhoeff circle and the Landolt broken ring. In recording the visual acuity, properly constructed letters of the alphabet, in my opinion, are the best clinical test objects available for literate subjects. For illiterates, the broken circle is useful, but is not so reliable as the 3 minute hook of Jackson. Why the latter has not been used more generally is difficult to understand. It answers every requirement of a visual test object. To comply with the requirements

for a proper test object for clinical purposes the width of the parts and the spaces between them—the details—should each subtend an angle of 1 minute, but there is no valid reason for assuming that the entire figure must subtend an angle of 5 minutes. Snellen himself did not follow this rule.

The test card should be evenly illuminated with a brightness ranging between 80 and 100 foot-candles, with fairly well lighted surroundings. With this amount of brightness small variations will have a negligible effect on the acuity of vision.

DR. F. H. VERHOEFF, Boston: This question of astigmatism is too difficult to discuss in the space of five minutes, so I shall simply state, in opposition to Dr. Beach, that I believe that astigmatic charts afford the best and most accurate means of determining astigmatism.

DR. WILLIAM H. CRISP, Denver: Concerning the question of illumination, it seems to me that one obtains important help from room illumination not applied directly to the test chart.

A KINETIC TEST FOR STEREOSCOPIC VISION. DR. F. H. VERHOEFF, Boston.

This paper will be published in full with the discussion in a later issue of the ARCHIVES.

LIPODYSTROPHIA PROGRESSIVA WITH OCULAR COMPLICATIONS. DR. J. W. CHARLES and DR. M. HAYWARD POST, St. Louis.

This paper was published in full in the *American Journal of Ophthalmology* (**19**: 126 [Feb.] 1936).

DISCUSSION

DR. MARTIN COHEN, New York: Of the numerous etiologic factors discussed, the most logical appears to be the trophoneurosis. This factor explains the local disturbance of fat metabolism, the symmetrical distribution and the abrupt line of demarcation. Trophoneurosis itself can be explained on the basis of localized scleroses of the trophic neurons. Such scleroses are now best understood to be caused by toxic substances, exogenous and endogenous. The second most logical etiologic factor is abiotrophy. In relation to the numerous other conditions in which disturbed fat metabolism is known to be the outstanding feature, this condition does not include all the symptom complexes.

In this case analyses of the blood and determinations of the basal metabolism and respiratory quotients have revealed nothing abnormal. These determinations of metabolism were made during the fasting state. The normal findings reported for this case do not exclude the possibility of abnormalities of metabolism of either carbohydrate or fat. Dr. DuBois has recently emphasized the practical value of making determinations of what he considers the total metabolism instead of determining the basal metabolism in the fasting state. Determinations of total metabolism include estimations of the patient's metabolic reactions, that is, his tolerance for and utilization of carbohydrates and proteins. This can be done most satisfactorily by ascertaining the changes in those constituents of blood and urine which are related to the metabo-

lism of fats and carbohydrates, also the changes in the total heat production and respiratory quotient as well as the fractionation of the calories between fats and carbohydrates after standard meals of carbohydrate and fat. When interpreted in terms of grams of carbohydrate or of fat metabolism per unit of body weight the results may indicate an abnormality of metabolism which is not demonstrable in tests made during the fasting state.

HEREDITARY MACULAR DEGENERATIONS. DR. RALPH I. LLOYD, Brooklyn.

From time to time various unusual conditions of the fundus have been described, some evidently hereditary and some isolated; some of the patients have had good and others very poor vision. There have been very few microscopic examinations of the choroid and retina to aid in classifying the various types. It is the consensus that these conditions should be assembled in one large group including those formerly described as choroiditis guttata (Tay), honeycomb choroiditis (Doyne), maculocerebral degeneration (familial) (Oatman), familial congenital macular degeneration (Best), progressive macular degeneration (Batten and Stargardt), tapetoretinal degeneration (Leber), retinitis punctata albescens (Gayet) and amaurotic familial idiocy (Tay and Sachs). The last is perhaps the most typical and best studied of the entire group. This type will be omitted from the present study.

Recently the senile and presenile macular degenerations have been added. There seem to be critical periods for each family group when the lesions appear and, as expected, the earlier they appear, the more severe they are. When they are severe, associated changes in the nervous system are not uncommon. The diagnostic features are: the significant family history and heredity, the bilaterality of the changes, the familial character of the lesions, which are alike in nature and time of appearance in the affected members of the family, and the uniform rate of progress and manner of evolution of the lesions in the affected members of the family.

DISCUSSION

DR. PARKER HEATH, Detroit: The most beneficial and the most enlightening information has come as the result of experimental studies. The respiratory membranes of Bowman and Bruch can be influenced by diets high in fat, alone and with such chemicals as phosphorus, so that one can produce these lesions at will. The early cell infiltrations are so different from the late scars that observers in this field have been misled. With increase in knowledge the nomenclature has been simplified, and the elaborate series of complicated processes have come to be termed "lipid cellular infiltrations." Acute familial diseases such as Niemann-Pick disease, Tay-Sachs disease or Gaucher's disease are thoroughly established as primary lipid diseases. Those which occur later, e. g., the juvenile, adolescent and senile changes, are probably the result of secondary influences such as those of endocrine imbalance, focal infections, insults from diet, and a multitude of other medical possibilities.

DR. GEORGIANA DVORAK-THEOBALD, Oak Park, Ill.: Little is known concerning the histologic changes in hereditary macular degeneration.

There are colloid excrescences on Bruch's membrane. These are in the form of half-circles or are flat and are covered by the pigmented epithelium. As they grow larger they may become devoid of pigment at the apexes. Owing to their light color and the thinning of the pigmented epithelium, they appear ophthalmoscopically as white or yellow dots. These dots are rarely encircled by pigment.

DR. SANFORD R. GIFFORD, Chicago: When my associates and I were looking over the children with this condition at a school for the blind in Illinois two or three years ago, we found several who had been blind almost from infancy, in whom the condition resembled retinitis pigmentosa. Within the last two months I have seen an almost blind infant with the changes of retinitis pigmentosa in the retinal periphery whose history was that of being almost blind from birth. So there are cases in which the condition has advanced to this stage in the first year or two of life.

Dr. Lloyd showed a picture of an older person with white spots about the macular region and degeneration of the macula. It seems to me that this may resemble a condition I have seen which is perhaps not generally recognized, in which the drusen of the lamina vitrea may be crowded so closely together in the macular region as to cause atrophy of the overlying retina. In nearly every case that I have encountered the condition had been diagnosed elsewhere as central choroidoretinitis, but I am sure there is a condition in which the drusen are so closely crowded that in older persons they may cause atrophy and a reduction of vision that is less than one would expect from the appearance of the fundus. One sees a considerable amount of pigment displacement, and the vision may be reduced to 6/21 or 6/15 but not to 6/60 as in cases in which there are productive changes in the macula.

In cases of familial macular disease without cerebral change I have had great success with the use of Zeiss' telescopic distal glasses.

DR. F. H. VERHOEFF, Boston: I have examined many eyes showing senile central macular changes. Knowing the various changes the pigmented epithelium can undergo, one can reconstruct ophthalmoscopic pictures from them and thus obtain a conception as to the histologic character of the macular lesions that are hereditary. The pigmented epithelium can undergo various alterations that are really degenerative even when they are due to hyperplasia, just as senile keratosis is degenerative although it is due to hyperplasia. In the senile form colloid excrescences are common. The result from proliferation of the pigmented epithelium with the formation of a so-called colloid substance which, as Coats pointed out, is similar to that of the inner layer of Bruch's membrane. Colloid excrescences may become calcified or organized. Instead of colloid excrescences, irregular proliferation may take place, some cells being heaped up and others being without pigment, and the formation of tissue similar to connective tissue may be noted. As mentioned by Dr. Heath, pigment cells loaded with fat deposits may also be found. The various changes that the pigmented epithelium may undergo are too numerous to be described in detail here.

In regard to the point brought out concerning the membrane of Bruch, this membrane consists of more than elastic tissue. A number of years ago I observed that the elastic layer in specimens stained with

hematoxylin often showed little patches of thickening. Ordinarily the elastic membrane does not stain with hematoxylin, but in hereditary macular degeneration one finds deeply stained spots in the membrane, which closely resemble nuclei, and several years ago, with Dr. Sisson, I went over my entire collection to ascertain how often this change occurred, and what its significance was. We found it to be very frequent, and the only correlation we could discover was with age. It had no significance as regards the visual acuity, and therefore I do not believe that Treacher Collins was correct in attaching any importance to it. I am very skeptical as to whether this change in the elastic membrane is ophthalmoscopically visible, because if it were, the spots would be much more commonly observed.

DR. S. JUDD BEACH, Portland, Me.: Dr. Lloyd's comprehensive paper affords me an opportunity to describe to the society the termination of 2 cases that I reported here ten years ago. These were cases of Oatman's maculocerebral degeneration, and they were remarkable as an exception to the rule that only in the cases which occur early is the degeneration severe. It will be recalled that Oatman's early cases, beginning at the second dentition, were the ones in which the condition progressed to dementia and death, whereas in the late group, beginning at puberty, although the appearances of the fundi were identical, the patients did not manifest mental disturbance.

My cases were also unique in that they occurred in a half-brother and half-sister related through the mother. The fathers were not related, and each had had perfectly normal children by previous marriages. The symptoms developed at puberty, that is, late. Yet when I first examined the children they had begun to deteriorate mentally just as did the patients in the early group. When I reported the cases, both patients had had convulsions, but neither had become completely antisocial. One patient was able to find employment in a dentist's office, and the other, although regarded as "odd," was working for the city as a day laborer. In 1933, six or seven years later, although the patients were nearly ten years apart in age, they simultaneously began to grow imbecilic. The older patient, the boy, must have been about 39 years old, and the younger, the girl, about 30. In the course of a year they became bedridden and finally died. I wish to report this termination, because it is a marked exception to the rule that in the later cases mental symptoms are not observed.

BULLOUS KERATITIS: A RATIONAL THERAPY. DR. JOHN GREEN, St. Louis.

This paper was published in full in the *American Journal of Ophthalmology* (**19**: 16 [Jan.] 1936).

DISCUSSION

DR. WILLIAM ZENTMAYER, Philadelphia: In 1930, before the College of Physicians, I presented a case of relapsing bullous keratitis in which an apparent cure was obtained by iridectomy. The patient had been faithful in giving attention to the eye and was regular in attendance at a clinic for six months, where the usual procedures, including irradiation and the use of the thermophore, were followed, without benefit.

There was zonular striping of the anterior capsule of the lens, and there had been temporary slight increase in the tension.

In November 1932, I presented the same patient after I had performed an iridectomy on the fellow eye because of an intractable relapsing bullous keratitis. Again the operation had resulted in a cure. There was neither increased tension nor zonular lamellae. The corneas showed marked endothelial dystrophy.

It has been discovered that the use of insulin in very small doses in the treatment of corneal dystrophy and even in that of ulcus serpens or corneal abscess gives astounding results. Although bullous keratitis such as Dr. Green has been discussing requires a little longer time to heal than the ordinary corneal abscess, it does respond to treatment. I have been stripping the cornea of all its superficial layers, and once or twice I have had success with chemical cauterization; however, one endeavors to solve this problem in accordance with the old theories and makes no progress. When one can restore the nutrition of the cornea to what is physiologically normal, one will be able to obtain cures in these cases.

Dr. Thomas D. Allen, Chicago: In my study of the subject I found that nature endeavors to fill in defects in Bowman's membrane by an extension of the epithelium and by production of connective tissue. At times the connective tissue becomes stratified, and the scar tissue becomes less opaque. One knows how the scars of ophthalmia neonatorum may all but disappear. Possibly this is the reason why another Bowman's membrane may very exceptionally appear. Undoubtedly there is a bulla, and if the bulla is due to an abnormality of Bowman's membrane, this upper membrane must be another Bowman's membrane, for the first Bowman's membrane is clearly followed deep into the substantia propria.

As a result of my study it seemed to me that the logical treatment for bullous keratitis is the removal of the offending tissue. When I removed Bowman's membrane in such a case I was gratified by the result. Certainly the result was far superior to that obtained by the simple removal of the epithelium and the scraping of Bowman's membrane or the application of tincture of iodine to it. Rereading Bowman's original article, I wondered if he did not actually remove some of the membrane as he states he did. He describes his results in these words: "After the removal of the corneal tissue I noticed that a more rapid complete healing of this eye followed than in the second quite similar eye on which I made no surgical intervention. I have, therefore, concluded that what I did at first only to make an examination was really a therapeutic procedure in such an unyielding disease. It ended in a complete transparent regeneration . . . ."

Dr. Derrick Vail, Cincinnati: I should like to support the work of Dr. Green and Dr. Allen by reporting 3 cases in which bullous keratitis was cured or relieved by deep cauterization of the cornea and the placing of a conjunctival flap.

Dr. Sanford R. Gifford, Chicago: I should like to call your attention to what von Szily Sr. said a long time ago in regard to the treatment of recurrent erosion of the cornea. Recurrent erosion seems to belong in the group considered by Dr. Green, although the condition I refer to

does not occur in glaucomatous eyes. It occurs when a slight injury to the cornea has caused an ordinary erosion which, instead of healing promptly as such erosions usually do, shows a continuous breaking down of the epithelium with recurrent attacks of intense pain. Among the large number of simple erosions which are seen in Chicago each year there are about two of this recurrent type. There are intense pain and swelling of the lids. Examination with the slit lamp shows edema of the whole corneal epithelium. Von Szily Sr. found that the whole corneal epithelium was usually loose. Acute attacks of pain occur on awaking in the night or in the morning. Von Szily concluded that the epithelium was only loosely healed and that when the upper lid was opened the epithelium was detached because of its adhesion to the lid. His treatment consisted in removing all the loose epithelium, which often included the entire surface of the cornea, with fine forceps, painting the denuded area with fresh compound solution of chlorine N. F. and closing the eyelids for four days. After removing the loose epithelium I have used 15 per cent tri-chloro-acetic acid instead of compound solution of chlorine N. F. and have followed with paracentesis. I believe that the important thing is to keep the eyes quiet for four days, which allows the corneal epithelium to heal firmly before the patient opens his eyes. It is necessary to bandage both eyes. This procedure has almost always been successful and leaves the cornea with practically no scarring. Almost all the patients had had numerous recurrences of the erosion previously, but they remained free from recurrences after this treatment unless a new traumatism occurred. Four days has been chosen as the period in which to keep the eyes quiet because the epithelium seems to heal firmly in this time.

Dr. Arnold Knapp, New York: Dr. Green spoke of a report I made some years ago in regard to the treatment of bullous keratitis by the application of solution of silver nitrate. May I say that in a subsequent case this treatment did not succeed in curing the disease. The process did not heal until after the corneal epithelium was removed and the galvanocautery was passed over the cornea, approximating the blunt tip to within a millimeter of the corneal tissue. The same effect may perhaps be obtained by means of the Shahan instrument.

Dr. William Tarun, Baltimore: At the present time I have a patient in whom the lesions are symmetrical. When first seen, the lesions were about 2.5 mm. in diameter; the surface bulged considerably and contained a limpid fluid. It occurred to me that it was important to use something that possessed the action of both a caustic and an irritant. After removing the loose epithelium, I carefully applied a 20 per cent tincture of iodine and employed a pressure bandage for some time. I ordered the administration of cod liver oil. The left eye was cauterized only once; the right eye, three times. The former now has a slight opacity or nebula and is quiescent, while the latter still has an occasional small bulla. The patient is comfortable, though the vision in the quiescent eye is slightly reduced.

The actual cautery has always seemed to me to be too severe and to cause needless destruction of Bowman's membrane, resulting in a greater amount of opacity.

DR. CONRAD BERENS, New York: I hoped that the grenz ray would be mentioned, so that there would not be need for further discussion, but since no reference to it was made, it seems worth while to call attention to a method of treatment that may have value: One of my patients, Mr. R. S., aged 52, was affected with a bullous keratitis associated with chronic iridocyclitis, which was resistant to autogenous vaccines prepared from material removed from his foci of infection and to repeated removal of all the epithelium, followed by the application of a 3 per cent tincture of iodine. Curettage of Bowman's membrane and the application of iodine have on several occasions also proved ineffectual. The Shahan thermophore was used twice, with only temporary improvement.

The grenz rays, 1,200 roentgens in divided doses varying from 150 to 250 roentgens, were applied to the cornea of the right eye over a period of seven weeks. A current of 10 milliamperes, 11 kilovolts peak and a distance of 9 cm. were used, which afford the maximum penetration given by the machine. The ray was coned down to an area 12 mm. in diameter at the aperture. Although one small bulla remains, the improvement has been greater and more permanent on irradiation with the grenz rays than on treatment by any of the other methods.

EFFECT OF MYDRIATICS ON INTRA-OCULAR TENSION. DR. HARRY S. GRADLE, Chicago.

This paper was published in full in the *American Journal of Ophthalmology* (19: 37 [Jan.] 1936).

DISCUSSION

DR. EVERETT L. GOAR, Houston, Texas: So far as I know, intraocular pressure has never been measured accurately before and after mydriasis, with classification of the results by decades from adult life to extreme old age. Such a study is therefore of considerable value.

I have never seen acute glaucoma follow the use of a cycloplegic, but I have known of 2 authentic cases in which it did. In neither of these, however, was the eye properly managed. In adults I contract the pupils with physostigmine following the use of a cycloplegic in study of refraction.

Concerning tonometry, I am of the opinion that too much value has been placed on certain phases of it and that the results are likely to be misleading. Certain circumstances, such as the patient's moving the eye slightly or squeezing the eyelids together, or the foot-plate's not being placed in the exact center of the cornea, or the patient's looking to one side rather than directly upward, may easily account for several millimeters of difference in the measurements.

DR. JONAS S. FRIEDENWALD, Baltimore: The change in the mean pressure that comes with age seems too small to be significant. In 200 normal eyes I was unable to find any special trend of the mean pressure for any age group. The cases in which there is a rise of pressure after mydriasis seem to fall into three groups: First there is the group in which the pressure change is measured as less than 4 mm. of mercury. Dr. Gradle attributes such a rise to an error of measurement. I should

like to know in how many cases there is a fall of pressure after mydriasis, for the probable error of measurement is most likely as great when the pressure is going down as when it is going up. The second group is clearly that of cases of preglaucoma. As a diagnostic procedure for this condition, Dr. Gradle's method appears to be most satisfactory. Finally, there is the third and most puzzling group of cases in which the pressure rises more than 4 mm. after mydriasis but in which subsequent tests fail to indicate that glaucoma is imminent. Although I have no explanation to offer, I suggest a factor for consideration. What one measures with the tonometer is not the intra-ocular pressure but a combination of the effect of the intra-ocular pressure and that of the elasticity of the ocular coats in allowing the weight of the tonometer to indent the cornea. A high reading with the tonometer may be due to high intra-ocular pressure or to low elasticity of the coats of the eye. Most mydriatics have the property of contracting the blood vessels, and it may be, therefore, that an increase in the tonometric reading indicates merely a decrease in the elasticity of the vascular bed without any change in the intra-ocular pressure.

Dr. E. C. Ellett, Memphis, Tenn.: I should like to discuss the incidence of glaucoma following the use of cycloplegics. I have encountered this unfortunate occurrence twice: once in a patient of my own, and once in some one else's patient whom I examined immediately after the glaucoma developed. The first patient was a woman in the twenties who gave evidence of having had an interstitial keratitis in childhood. I used homatropine hydrobromide U. S. P. in the usual manner in the determination of her refraction, which showed a low degree of compound myopic astigmatism. Two days later glaucoma developed in one eye. She received treatment and was finally operated on. This occurred twenty-five years ago, and the eye has remained well, with normal vision, normal tension and normal visual fields. About a year ago acute glaucoma developed in the other eye. She was not under my care at the time, but she was successfully operated on and has recovered the use of the eye.

The second patient was a woman who gave a family history of glaucoma. I do not recall the exact details, but there were several cases of glaucoma in the family, and it was so firmly impressed on her mind that she mentioned it, and I therefore measured the error of refraction without the use of a cycloplegic. On a subsequent occasion she consulted another oculist, who used homatropine hydrobromide, which precipitated an attack of glaucoma in one eye. She was treated by medical measures, with complete recovery, but two or three years later she again had trouble in that eye and failed to see an ophthalmologist until the condition was well advanced, with the result that she lost the vision in that eye.

Dr. F. H. Verhoeff, Boston: It is commonly assumed that a shallow anterior chamber is indicative of approaching glaucoma, and I should therefore like to ask Dr. Gradle if he noted, in the cases in which he found an increase in pressure whether there was in most of them a shallow chamber, and also whether this was present in those cases in which he found a condition that was certainly preglaucomatous.

Dr. Harry S. Gradle, Chicago: The stippling of the cornea resulting from the use of the tonometer is a very disturbing element in refrac-

tion if the refraction is studied immediately afterward. The stippling persists for thirty minutes and then disappears. As the refraction is not studied until fully an hour after the tonometer has been applied, this stippling becomes unimportant.

The number of cases in which there was a fall in pressure at the second measurement was very small. I cannot give the exact figures, but the decline in pressure was less than the increase in pressure, so that the point raised by Dr. Friedenwald is possibly pertinent, namely, that the increase of from 2 to 4 mm. may not have been due entirely to a personal error but may have been due to the effect of the mydriatic.

As to the possibility that the contraction of the blood vessels within the eye was responsible for the increase, that does not sound logical to me. In the first place, none of the vasoconstricting effects of the mydriatics used would persist for that length of time even if the pupillary dilatation continued. The vasoconstrictor effect of epinephrine lasts from fifteen to twenty minutes at the maximum, and it is improbable that the effects of any of the preparations used for obtaining mydriasis would persist for a much longer time than that.

Dr. Ellett spoke of an acute uncompensated glaucoma that developed after the use of a mydriatic. None of my patients showed lack of compensation, but in 1,000 cases there were 9 of a condition definitely preglaucomatous, in which it could be proved by provocative tests that in the course of time positive glaucoma would develop. Secondly, I believe that this procedure is of value in preventing the development of glaucoma of the compensated type in the patient so predisposed. In other words this type can be diagnosed much earlier and, by adequate medicinal therapy, can be kept under control.

As to the absolute depth of the anterior chamber, no record was made in any case before the mydriatic was used.

TREATMENT OF SARCOMA OF THE UVEAL TRACT. DR. W. GORDON M. BYERS and DR. J. A. MACMILLAN, Montreal, Canada.

This paper was published in full in the December 1935 issue of the ARCHIVES, p. 967.

TUBERCULOSIS OF THE CHOROID ASSOCIATED WITH GENERALIZED MILIARY TUBERCULOSIS. DR. FREDERICK T. TOOKE, Montreal, Canada.

An infant presented miliary tuberculosis of the meninges, lungs, liver, spleen, kidneys and adrenal glands, tuberculosis of the right peribronchial gland, the thoracic and the abdominal lymph glands, exudative ulcerative tuberculous enteritis and a tuberculoma of the left cerebral hemisphere. Microscopically the choroid showed a more or less generalized miliary tuberculous process, included entirely within choroidal tissue, rather than true tubercle formation as revealed by the ophthalmoscope. The conditions in the pia arachnoid and in the vaginal space of the optic nerve with the negative findings elicited by lumbar puncture on admission substantiated the contention that tubercles of the choroid originate from the blood stream as perivascular tubercles. Tubercle bacilli were demonstrated in sections.

VITREOUS DETACHMENT, ANTERIOR DIALYSIS AND PRESENCE OF A TUMOR-LIKE MASS OVER THE OPTIC NERVE. DR. T. B. HOLLOWAY AND DR. W. E. FRY, Philadelphia.

An unusual vitreous and retinal detachment in a 22 year old patient who had suffered a trauma of the head is presented, and the microscopic examination of the specimen is fully reported.

DISCUSSION

DR. F. H. VERHOEFF, Boston: I have never observed clinically the condition which Dr. Holloway has described and have never recognized it microscopically. It is possible that I may have seen it microscopically and regarded it as due to shrinkage and imperfect sectioning. I note that in this case there is separation of the choroid. I should like to ask Dr. Holloway whether the tension of the eye was taken.

DR. T. B. HOLLOWAY, Philadelphia: The tension of the right eye was 23 mm. and that of the left eye 17 mm.

DR. F. H. VERHOEFF, Boston: The tension was a little low then. It seems to me, therefore, that the separation of the choroid may not have been an artefact. Artificial separation of the choroid is very unusual unless some membrane is exerting traction on it. The patient may have been blind for a considerable time before the condition was discovered; if so, one expects to find colloid excrescences and macrophages on the choroid, and these are not present. I agree with Dr. Holloway, therefore, that the blow on the head was probably the cause of the condition.

DR. T. B. HOLLOWAY, Philadelphia: I failed to show the slide depicting the section of the globe pertaining to Pagenstecher's case. The similarity to our case will be noted in that it shows the same mass of convolutions proceeding from the position of the disk.

THE CRYSTALLINE LENS: V. THE NATURE OF THE REDUCING SUBSTANCES IN THE LENS. DR. E. V. L. BROWN and E. I. EVANS, PH.D., Chicago.

This paper will be published in full in a later issue of the ARCHIVES.

STRUCTURE OF THE VITREOUS. DR. JONAS S. FRIEDENWALD and ROBERT D. STIEHLER, PH.D., Baltimore.

This paper was published in full in the November 1935 issue of the ARCHIVES, p. 789.

THE MECHANISM OF THE FOVEA. DR. FRANCIS H. ADLER and DR. GEORGE MEYER, Philadelphia.

Ideas of the function of the fovea have been dominated largely by the teachings of physiologic optics, which describe point-images accurately focused on individual cones. On this basis the acuity of vision must be limited by the size of the cones packed together in the fovea, the "grain" of the retina and the sharpness with which the dioptric system of the eye can focus an image on the cones. Because of the

aberrations from which every eye suffers, point-images cannot exist in any eye, even an emmetropic one, and it has been shown that even during the most careful fixation, movements of the eye and head occur which are too large to permit stimulation of individual cones by the images which even a perfect dioptric system can form. The acuity of the eye as measured by the alining power exceeds the possible "grain" of the retina.

The investigation discussed was undertaken to throw some light on how the foveal mechanism is capable of such fine discrimination. An instrument is described by which the alining power of the various portions of the retina can be tested while keeping the fixation of the eye, the state of adaptation of the retina and the size of the pupil constant. It was found that within a comparatively large area of the macula the threshold of the alining power is at a maximum and does not vary, but that outside this area, which is sharply delimited, there is a sudden drop in acuity. This area is spoken of as the physiologic fovea. The factors which influence the acuity of this area are discussed, and a tentative theory of the mechanism by which such low thresholds are obtained is presented.

CATARACTS PRODUCED IN ALBINO RATS ON A RATION CONTAINING A HIGH PROPORTION OF LACTOSE OR OF GALACTOSE. DR. ARTHUR M. YUDKIN and CAROLINE H. ARNOLD, A.B., New Haven, Conn.

This paper was published in full in the December 1935 issue of the ARCHIVES, p. 960.

SEROUS RETINITIS. DR. BERNARD SAMUELS, New York.

The so-called serous iritis is reviewed briefly, and an identical and parallel disease in the retina, named serous retinitis, is described. The two diseases are independent of each other. The paper is restricted to a consideration of injured eyes in which the lens had been greatly damaged. The lesion, whether in the iris or in the retina, is attributed to the action of irritating substances derived from free lens matter.

The following interpretation of the clinical aspects of serous retinitis is based on the pathologic observations:

The diagnosis of atrophy of the ocular globe so often made was not correct. Some of the eyes may have exhibited hypotension, but this was due to infiltration of the ciliary body by lymphocytes, just as in an ordinary case of iridocyclitis. Some of the eyeballs showed signs that the inflammation was subsiding, and it seems that a few of them might have been saved from enucleation. The essential symptoms of atrophy of the globe, namely, membranes on the flat part of the ciliary body and detachment of the retina, were not present. It is unlikely that the condition in any of the eyes would have gone on to actual shrinkage.

The failing vision was due not to a detachment of the retina but to a characteristic process in its stroma and in the papilla.

The reason assigned for the removal of an eye in most of these cases, namely, fear of sympathetic ophthalmia in the fellow eye, was justifiable in certain cases inasmuch as, of 59 eyes, 6, that is, about 10 per cent, revealed the specific sympathetic infiltration in the choroid.

DISCUSSION

Dr. Arnold Knapp, New York: Dr. Samuels speaks of serous retinitis and then of a condition that is due to severe injury of the lens. I do not believe that these changes go well together. All are familiar with the results of a severe injury to the anterior part of the eye which is followed by changes in the optic nerve and greatly reduced vision, this reduction in vision presumably being due to involvement of the macula or of the papillomacular bundle. I am going to ask Dr. Samuels if he will be good enough to say whether, in his histologic examination, he has found any anatomic explanation for the central scotoma.

Dr. Bernard Samuels, New York: Neither the iris nor the ciliary body was ever very severely damaged. In almost every case there was great damage to the lens, and in a number of cases the perforation extended through the lens into the vitreous, so that it was concluded that the reaction in the iris and the retina was due to absorption of toxins from lens material. The choroid did not show changes, apparently for the reason that it is protected from toxins by the retina.

In explanation of the central scotoma, the macular region of the retina was particularly involved in the reaction, and the papilla also participated. The principal seat of the process in the retina was in the posterior part of the globe. In most instances the nerve trunk appeared to be normal posterior to the lamina cribrosa sclerae.

Epithelial Downgrowth into the Anterior Chamber Following Cataract Extraction: Arrested by Radium Treatment. Dr. Derrick Vail, Cincinnati.

This paper was published in full in the February 1936 issue of the Archives, p. 270.

Detachment of the Choroid After Cataract Extraction: Clinical and Experimental Studies, with a Report of Seventy-Five Cases. Dr. C. S. O'Brien, Iowa City, Iowa.

This paper was published in full in the October 1935 issue of the Archives, p. 527.

Results of Cataract Extraction Five Years and More After Operation. Dr. E. C. Ellett, Memphis, Tenn.

One hundred and eighty patients were observed. Of this number 107 were operated on by capsulotomy and 73 by intracapsular extraction. The present condition of these patients, from five to thirty-five years after operation, is recorded, with comment on the late results.

An Analysis of One Thousand, Three Hundred and Forty-Three Intracapsular Cataract Extractions by Forty-Eight Operators Following the Verhoeff Method. Dr. Allen Greenwood, Boston, and Dr. Herman P. Grossman, Providence, R. I.

A statistical analysis of 1,343 attempted intracapsular cataract extractions by the Verhoeff method performed at the Massachusetts Eye and Ear Infirmary from 1927 to 1934 by the visiting and resident

staff shows 1,166 cataracts delivered in intact capsule (86.8 per cent) and 115 ruptured capsules delivered by forceps after extraction of the cataracts through the capsular rents (8.6 per cent). In 62 cases of attempted intracapsular extraction, the capsule was ruptured and not delivered (4.6 per cent). In this entire series, loss of vitreous occurred in 6.2 per cent. We conclude: 1. The Verhoeff intracapsular method is a safe technic to employ from the standpoint of the frequency of rupture of the capsule and of loss of vitreous. 2. If the capsule is accidentally ruptured, it is more conveniently and thoroughly removed in this procedure. 3. This process has definite theoretical advantages regarding the manipulation of the lens, these advantages being substantiated by our experience in 1,343 operations. 4. The Verhoeff method may be successfully employed for any cataract other than the juvenile or the traumatic one. 5. If the cataract is not permitted to progress to the stage of maturity or hypermaturity, extraction is safer and more successful. 6. Any operator of ordinary skill and experience in ocular surgical procedures need not hesitate to acquaint himself with this technic.

DISCUSSION OF PAPERS OF DR. E. C. ELLETT AND DRS. GREENWOOD AND GROSSMAN

Dr. John M. Wheeler, New York: Dr. Ellett gives the impression that glaucoma following extraction of a cataract usually yields to nonsurgical treatment and that if it does not it will yield readily to surgical treatment. To me, one of the most disturbing things following extraction of a cataract is ocular hypertension, because I do not feel confident that I can reduce the tension and have it remain normal after the operation for hypertension.

The paper by Dr. Greenwood and Dr. Grossman brings one two valuable points of information: The first is that the grasp of the capsule advocated by Verhoeff enables the surgeon to remove the lens and its capsule in a very high percentage of cases. In other words, the grasp of the capsule in its relatively thick portion at or near the equator gives a 95 per cent chance of extraction of the capsule. The second point is that in this series of cases with a large number of surgeons operating it was possible to remove the lens in the capsule with a very low percentage of losses of vitreous—a lower percentage than was obtained when extraction of the capsule was not in vogue. I believe that another point mentioned in the paper should be stressed, namely, that improved anesthesia of the globe was of some importance, and especially that the use of procaine hydrochloride for relaxation of the orbicularis oculi was of very great importance—greater perhaps than the authors realize, so that the previous series which was reported would have made a considerably better showing if the blepharospasm had been controlled by the use of this anesthetic.

Another point which is not mentioned in the paper but which I believe is of importance is that an iridectomy is presupposed. In other words, in practically all these cases the extractions were accompanied with or preceded by complete iridectomy. Some are of the opinion that there is a decided advantage to the patient in having an active round pupil following extraction of a cataract as well as previous to it,

and for those who wish their patients to have active round pupils this operation would not be suitable. Another technic would have to be selected.

DR. H. J. HOWARD, St. Louis: I am sorry that the authors did not bring out some of the other features of the Verhoeff method, such as the use of a special forceps and a scleroconjunctival suture, because in my opinion the results obtained at the Massachusetts Eye and Ear Infirmary and in other institutions using the Verhoeff method have to do with, and are dependent on, the technic as a whole.

In China during the period from 1919 to 1927 I demonstrated Dr. Verhoeff's method to 160 doctors, most of them Chinese, the others American and British, and taught the method in detail to 11 Chinese ophthalmologists. I believed that this method was the safest one to teach and that the results obtained were at all times good, even in the hands of novices.

I do not have the records of the operations done in China or of those performed in this country except those of the past two years. Of the 60 cases put on record during the latter period in which Dr. Verhoeff's intracapsular method was attempted, the capsules in 5 ruptured and the lenses were removed extracapsularly. There was loss of vitreous in 3 cases, 2 being cases of complicated cataract. These results seem to parallel somewhat closely those reported by Dr. Greenwood and Dr. Grossman.

DR. H. P. GROSSMAN, Providence, R. I: In answer to Dr. Wheeler regarding the results obtained in this series, I wish to say that it is impossible, in a short paper of this type, to enter into any lengthy discussion of ultimate results. I feel, however, that if one desires to deliver lenses in capsules, if one does so with a low incidence of loss of vitreous and without increase in intercurrent complications, and if, after all that, the results obtained are not satisfactory, there must be other conditions present besides cataracts.

With reference to Dr. Wheeler's point concerning the necessity of performing an iridectomy when using the Verhoeff method: Of late the extraction with a round pupil has been done in a small number of cases. I have performed it in a few cases myself, and many of the younger men have been able to employ the Verhoeff method without an iridectomy. This has been accomplished by means of a special forceps, which has a heel on the back of the curve for the purpose of retracting the iris before the capsule is grasped. The remainder of the operation follows precisely the routine Verhoeff technic.

EFFECT OF BACTERIAL LYSATE ON STAPHYLOCOCCIC KERATOCONJUNCTIVITIS IN RABBITS. DR. MAX M. STRUMIA and DR. HUNTER W. SCARLETT, Philadelphia.

This paper was published in full in the January 1936 issue of the ARCHIVES, p. 47.

INCLUSION BODIES IN OPHTHALMIA NEONATORUM: FURTHER NOTE. DR. SANFORD R. GIFFORD and DR. NATHAN K. LAZAR, Chicago.

This paper was published in full in the August 1935 issue of the ARCHIVES, p. 197.

METASTATIC OPHTHALMIA IN A PATIENT WITH PNEUMONIA: BACTERIOLOGIC OBSERVATIONS. DR. S. HANFORD MCKEE, Montreal, Canada.

Among the micro-organisms producing metastatic ophthalmia the streptococcus ranks first, followed closely by the pneumococcus. Other micro-organisms specific for certain diseases, such as the meningococcus, the pneumobacillus, the influenza bacillus and the typhoid bacillus, are also found. The meningococcic and pneumococcic conditions are mild with comparative frequency. Metastatic ophthalmia occurs chiefly in association with pyemia and, in rare instances, with such acute infectious diseases as typhoid, typhus, variola, scarlet fever, anthrax, influenza, ulcerative endocarditis, diphtheria, erysipelas, Weil's disease and pneumonia.

A fatal case of lobar pneumonia caused by pneumococcus type I is presented, in which this pneumococcus was demonstrated in connection with an associated metastatic ophthalmia.

DISCUSSION

DR. GEORGIANA DVORAK-THEOBALD, Oak Park, Ill.: Sequini made a thorough search of the world's literature on this subject up to the end of 1922 and found that 342 cases had been reported. In only 106 of these was a bacteriologic examination made. The cocci predominated.

In 1933 Lumbroso searched the literature for reports of cases occurring during the ten years following Sequini's report. He found 14 instances in which metastatic ophthalmia followed puerperal fever, 12 in which it occurred after surgical infections and 65 in which it followed generalized infections. In only 7 of these 65 cases did the ocular disease come in the wake of pneumonia or bronchopneumonia. In 7 cases it followed influenza and the pneumococcus was isolated from the blood. According to many authors, the retina is the most frequent site of endocular infection; next comes the choroid.

Lawson pointed out that when bacteria enter the vitreous they multiply abundantly in it but do not ordinarily pass into the coats of the eye. The purulent inflammation depends, therefore, on the remote action of the bacteria. The inflammation affects mainly the inner surface of the membranes lining the vitreous, producing endophthalmitis. Necrosis of the coats of the eye develops when the bacteria are abundant or virulent.

DR. ALLEN GREENWOOD, Boston: Several years ago I reported that in my service during the World War I observed 3 cases of metastatic choroiditis resulting in panophthalmitis in solidiers each of whom had a compound fracture of the femur. These were the only cases of metastatic choroiditis that I saw during a long service with the British and American troops.

DR. H. J. HOWARD, St. Louis: I want to add to the records that of a case which I had a few weeks ago. A woman, about 35 years of age, had for several weeks a low grade influenza followed by secondary otitis media associated with purulent blisters on one palm and one heel. She next presented metastatic endophthalmitis in one eye, involving only the posterior segment. The eye was lost. Bacteriologic studies were made, and the pneumococcus was found in pure culture. The patient survived and is now in good health.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Nov. 21, 1935

J. MILTON GRISCOM, M.D., *Chairman*

ALEXANDER G. FEWELL, M.D., *Clerk*

TWO CASES OF BLOOD-STAINING OF THE CORNEA. DR. T. B. HOLLOWAY.

A boy aged 10 years was struck and injured in the left eye by a stone. Twenty-four hours later the eye showed iridodialysis and a small hemorrhage in the anterior chamber. On the following day the hemorrhage had increased. Sixteen days after the injury a paracentesis and irrigation of the anterior chamber were done. Five weeks after the injury the boy had typical blood-staining of the left cornea, and the vision was reduced to perception of light. One year after the accident there was hypotonus but no blood-staining of the cornea.

A girl aged 11 years was struck on the left eye by a stone. Pain followed for about an hour, but there was no apparent loss of vision. During the evening of the second day severe pain developed in the affected eye, with blindness. On the sixth day vision was reduced to perception of light, the anterior chamber was completely filled with blood, and the tension tested by finger was plus 2. On the eleventh day slight evidence of blood-staining of the cornea was observed. This increased rapidly so that within three days the whole cornea was deeply stained. No immediate paracentesis was done because of some confusion pertaining to the cultures—it was thought there was a streptococcic contamination. With slit-lamp illumination the usual appearance of fine granules deep in the cornea was noted, most marked centrally. Later a paracentesis was done with slow evacuation of the contents of the anterior chamber, which was followed by clearing of the peripheral region of the cornea.

AN UNUSUAL CASE OF BLOOD-STAINING OF THE CORNEA. DR. WARREN S. REESE.

In a case of blood-staining of the cornea, Dr. Beideman did a combined cataract extraction in May 1935. In August, following a capsulotomy, an effusion of blood into the corneal lamellae appeared a few millimeters from the incision and gradually extended until it occupied an oval area in the upper nasal quadrant about 4.5 mm. wide and 5.5 mm. long. Since then this area has changed little. Notwithstanding the corneal lesion, the visual acuity when corrected was 6/6.

DISCUSSION

DR. ALFRED COWAN: Blood-staining of the cornea is much more common than the literature indicates. I have seen 3 instances of blood-staining of the cornea at the Wills Hospital within the past two weeks. There is always a greater deposit of granules in the central portion of the cornea, arranged so densely that the gross appearance is that of a

brownish disk in the aqueous. The corneal staining does not seem to depend on permeability of the endothelium. I have never found a break in the endothelium to give rise to absorption of aqueous by the cornea. One sees any number of cases in which the endothelium is broken and in some instances entirely absent while the cornea remains perfectly clear. The periphery of the blood-stained cornea is comparatively clear, although there are some granules deposited there also.

The condition described by Dr. Reese is not true blood-staining of the cornea, but a thin layer of blood lying in a plane which extends in from the limbus above and about half-way in the thickness of the cornea. It is probably a hemorrhage between the lamellae which has been sealed off and retains its bright red color.

SYMBLEPHARON FROM PEMPHIGUS CORRECTED WITH MUCOUS MEMBRANE GRAFT. DR. EDMUND B. SPAETH.

A case was presented showing the results of operative treatment of a symblepharon which originated from pemphigus. There had been complete loss of the lower culdesac with partial loss of the upper one, especially at the canthal angles. Moving pictures of this operation were shown at the College of Physicians of Philadelphia earlier this year. The results at the time of the meeting were shown to emphasize the value of a mucous membrane graft as compared with an epithelial one for this type of defect.

HISTOPATHOLOGY OF CHOKED DISK. DR. BERNARD SAMUELS.

Preparations of edematous nerve heads taken from fifty eyeballs from cadavers form the basis of this study. The diagnosis of papilledema had been made in each instance in life. In 14 cases the cause of the edema was given in life as tumor of the brain, and in the remaining 31 cases various causes were assigned, such as nephritis, meningitis, exophthalmos and anemia. It was possible in the sections to differentiate between the recent and old cases. In the recent cases there was only a separation of the nerve fibers by fluid, whereas the old cases were marked by atrophy of the ganglion cell layer of the retina and of the nerve bundles and proliferation of the endothelial cells of the capillaries and of the glia cells in the papilla. The tendency toward circumpapillary detachment of the retina was discussed. In measuring the swelling in the anatomic preparations the diameter of the sclerochoroidal canal was taken as a standard for estimating the diameter of the swelling, and the thickness of the edematous disk was spoken of as being equal to, or so many times greater than, that of the retina. The effect of the swelling of the nerve head on the retina as a whole and on its different layers was minutely gone into, as this was one of the chief purposes of the demonstration. A peculiar triangular tissue that appeared mostly on the nasal side of the regular neuritic swelling, sometimes containing blood vessels, was discussed in detail. Changes in the pigmented epithelium were noted, and remarks were made on what sometimes appeared to be a hernia of the nerve fibers interposed between the pigmented epithelium and the retina. No outspoken inflammatory reaction was noted in any of the papillae, no mat-

ter what cause was ascribed for the swelling. In so large a material, it was of interest that the intravaginal hydrops seemed to bear no quantitative relationship to the swelling of the papilla. It was found that extreme edema was not always associated with marked hydrops. An attempt was made to correlate the histologic observations with the clinical aspects of papilledema.

Book Reviews

Outlines of Ophthalmology. Parts I and III. By D. E. Rolf, M.D. Price, 50 cents. Pp. 52. Chicago: Division of Ophthalmology, University of Chicago, 1935.

This outline was published with the aid of a grant from the Kramer Fund. Part I deals with anatomy, histology, physiology, muscles and optics, while Part III deals with the treatment of some of the more common diseases of the eye. (Part II, which is to appear later, will deal with diagnosis, pathology, bacteriology and perimetry.)

The first sixteen pages are concerned with the gross, microscopic and regional anatomy of the eye, orbit and orbital contents. For one who has already studied these subjects in one of the standard textbooks, this section will furnish a comprehensive and condensed résumé of the pertinent facts.

The three pages devoted to the intra-ocular fluid include a detailed outline of the factors by which the normal intra-ocular tension is maintained as well as the factors by which it may be varied.

Three pages are allotted to the pupils; these are devoted to their innervation and pharmacology and the normal and abnormal pupillary reactions which occur in health and in disease. In this section Rolf might, with advantage, have included notes concerning the application of the new humoral physiology to the mechanism of pupillary constriction and the now generally accepted theory as to the mechanism by which physostigmine acts as a miotic.

Elementary optics and refraction are given eight pages. The author does not clearly define astigmatism "with the rule" and "against the rule." Also, in mentioning the treatment of presbyopia, he states that a lens of from + 1.00 to + 3.00 diopters is added to the distant glass for near work, which is inaccurate.

Five pages are devoted to the extra-ocular muscles, heterophoria and nystagmus. In outlining the muscles used in carrying out the oblique conjugate movements of the eyes, the author has apparently followed the British custom of referring to the muscles used by the patient in looking to the *examiner's* right or left, rather than to the muscles used in looking to the *patient's* right or left. This is likely to be a source of confusion.

The remaining nine pages of Part I deal with color vision, color blindness, theories of vision and retinoscopy. In his discussion of retinoscopy Rolf does not directly correlate the movements of the retinal image with the movements of the mirror. While his intentions are obvious, his explanation might be confusing to a beginner.

In Part III, which is devoted to therapy, the instructions are brief, concise and fundamentally sane.

Part I of this outline is obviously an abbreviated synopsis of a well known textbook of ophthalmology, as it follows rather uniformly the arrangement of this textbook.

In the reviewer's opinion, this outline will be of greatest value to those who are alerady well acquainted with the basic elements of ophthalmology and who desire such an epitome for rapid reference or review. It will not replace a textbook for either interns or students, but may be of value in preparing for examinations.

Copies are obtainable from the trustees of the Kramer Fund, Dr. E. V. L. Brown and Dr. L. Bothman, 122 South Michigan Avenue, Chicago.

W. F. DUGGAN.

Twenty-Second Annual Report of the Ophthalmic Hospitals Section for 1934. Price, 12 piasters. Pp. 46. Cairo: Government Press. 1935.

During the year of this report a number of new units have been added to the ophthalmic hospitals in Egypt. The clinical work for 1934 was as follows: new patients, 928,215; inpatients, 27,860, and operations, 305,206—an increase of nearly 10 per cent over the figures for 1933. The number of patients found to be blind in one or both eyes was 5.9 per cent. The causes for this blindness are given in a special table. Acute ophthalmia was the cause in 81 per cent of the cases, and the gonococcus was the predominating factor of infection. Glaucoma was the cause of blindness in 10 per cent of the cases.

The number of operations performed during 1934 was 305,206. Of these, 1,700 were for senile cataract, 370 for soft cataract and 89,674 for trichiasis and entropion. Other operations numbered 27,067. The Snellen operation for trichiasis and entropion was the one generally practiced. Extraction for cataract was the usual extracapsular operation with iridectomy. There were 1,297 trephinings, and 11 operations for detachment of the retina.

A brief report by the director of the Memorial Ophthalmic Laboratory at Giza shows that 781 examinations were made.

Many statistical tables are added, and two charts conclude the report.

The report also draws attention to the fact that the Fifteenth International Ophthalmic Congress is to be held at Cairo in December 1937. Dr. Maziny Bey, who is the director of the ophthalmic hospitals in Egypt, has been elected technical president of the congress. The subjects for discussion are to be (*a*) "Endocrinology and the Eye" and (*b*) "Hypertension and Retina." The subscription is fixed at 50 gold Swiss francs. An Egyptian local committee will have charge of the organization and the necessary arrangements for the coming congress.

ARNOLD KNAPP.

Tabulae Pseudo-Isochromaticae. By C. G. Bostrom, M.D. Pp. 16. Stockholm, Sweden: Kifa, 1935.

This book contains sixteen pseudo-isochromatic plates for testing color vision, together with a small pamphlet of directions for their use.

The series consists of the following: (1) Two simulator plates. A person who, provided his vision with or without glasses is 0.3 or better, says that he cannot read these plates is a simulator (of color

blindness). (2) Ten plates, all of which should be decipherable by persons with normal color vision. (3) Four dissimulator plates. These plates, which resemble superficially the preceding ten plates, have no figures. A person who points out certain figures on these plates is a dissimulator.

The plates are to be used preferably in full daylight, and each should be read within fifteen seconds. Plates III, IV, VII and VIII are for detecting deuteranopia, and plates V and VI are for detecting protanopia. Plates IX to XII, which are extremely difficult, are illegible not only to protanopes and deuteranopes but also to those with a middle degree of anomaly.

These plates are well made and should serve as a useful adjunct to the well known Ishihara plates.

W. F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: December 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Oxford. Time: July 22-24, 1936.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. R. Foster Moore, 53, Harley St., London, W. I.
Secretary: Miss Ida Mann, 7, Wimpole St., London, W. I.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: July 6-8, 1936.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. John Green, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Kansas City, Mo.: Time: May 11-15, 1936.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: New York. Time: Sept. 26 to Oct. 3, 1936.

American Ophthalmological Society

President: Dr. L. S. Greene, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 1-3, 1936.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston, Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. H. G. Merrill, 3245 4th St., San Diego, Calif.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Del Monte, Calif. Time: Spring, 1936.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.
Place: Seattle. Time: January 1937.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. W. R. Fringer, 228 S. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. George B. Garlick, 144 Golden Hill St., Bridgeport.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.
Place: Savannah. Time: May 12-15, 1936.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Edwin Cobb, 307 Masonic Temple, Marshalltown.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Marshalltown. Time: September 1936.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. E. Binger, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Place: St. Paul. Time: May 1936.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. George A. Lewis, Roundup.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Secretary: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Burton W. Fassett, Geer Bldg., Durham.
Secretary-Treasurer: Dr. Casper W. Jennings, 332 N. Elm St., Greensboro.
Place: Durham. Time: October 1936.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.
Place: Jamestown. Time: May 1936.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Emory Hill, 501 E. Franklin St., Richmond.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
Place: Richmond. Time: May 1936.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William O. Martin Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. A. V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Robert B. Blue, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. F. W. Thomas, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. H. Newton, Mercantile Bldg., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich
Place: Various local hospitals. Time: Third Thursday of alternating months September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Clyde E. Harner, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul T. Southgate, 704 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter R. Crane, 756 S. Broadway, Los Angeles
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Hilmar G. Martin, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in alphabetical order.
Secretary: Dr. C. W. Rucker, 527 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. W. J. Blackburn, 663 Reibold Bldg., Dayton, O.
Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Kate S. Zerfoss, 119, 7th Ave., N., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St.,, New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. John H. Judd, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassidy, 107 S. 17th St., Omaha
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. E. C. Reynolds, 657 Main Ave., Passaic, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Robert H. Courtney, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Meyer Wiener, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. D. Brazeau, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. J. B. Stone, 1105 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Arnold B. Kauffman, 104 W. Onondaga St., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St. W., Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.